WEST Search History

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DATE: Monday, January 17, 2005

Hide?	Set Name	Query	Hit Count
		DB=USPT; PLUR=YES; OP=OR	
☐	L7	L6 same (IL-12 or interleukin-12)	14
☐	L6	(inhibit or scavenge) with (NO or nitric adj oxide)	15878
		DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=OR	
☐	L5	L4 and (IL-12 or interleukin-12)	45
☐	L4	monomethyl adj arginine or (l-NMMA)	424
		DB=USPT; PLUR=YES; OP=OR	
☐	L3	L2 and (IL-12 or interleukin-12)	4
☐	L2	monomethyl adj arginine	63
☐	L1	monomethyl adj arginine with inhibitor	19

END OF SEARCH HISTORY

FILE 'CAPLUS' ENTERED AT 18:21:44 ON 17 JAN 2005
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=> s (Il-12 or interleukin-12) (a) (nitric (w) oxide (w) inhibit or nitric (w) oxide (w) neutraliz?)
L1 0 (IL-12 OR INTERLEUKIN-12) (A) (NITRIC (W) OXIDE (W) INHIBIT OR NITRIC (W) OXIDE (W) NEUTRALIZ?)

=> s (Il-12 or interleukin-12) and (nitric (w) oxide (w) inhibit or nitric (w) oxide (w) neutraliz?)
L2 3 (IL-12 OR INTERLEUKIN-12) AND (NITRIC (W) OXIDE (W) INHIBIT OR NITRIC (W) OXIDE (W) NEUTRALIZ?)

=> duplicare remove l2
DUPLICARE IS NOT A RECOGNIZED COMMAND
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=> duplicate remove l2
DUPLICATE PREFERENCE IS 'CAPLUS, USPATFULL'
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PROCESSING COMPLETED FOR L2
L3 3 DUPLICATE REMOVE L2 (0 DUPLICATES REMOVED)

=> d l2ibib, abs
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'L14' IS NOT A VALID FORMAT
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'D' IS NOT A VALID FORMAT
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individual files.
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L3 ANSWER 1 OF 3 USPATFULL on STN
ACCESSION NUMBER: 2002:156684 USPATFULL
TITLE: Methods and compositions for enhancing the
immunostimulatory effect of ***interleukin*** - ***12***

INVENTOR(S): Trinchieri, Giorgio, Charly, FRANCE
Lee, William M. F., Wynnewood, PA, UNITED STATES
Koblish, Holly, Yardley, PA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002081277	A1	20020627
APPLICATION INFO.:	US 2002-79068	A1	20020220 (10)

RELATED APPLN. INFO.: Division of Ser. No. US 1999-395038, filed on 13 Sep 1999, GRANTED, Pat. No. US 6375944

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-101698P	19980925 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HOWSON AND HOWSON, ONE SPRING HOUSE CORPORATION CENTER, BOX 457, 321 NORRISTOWN ROAD, SPRING HOUSE, PA, 19477
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 7 Drawing Page(s)
LINE COUNT: 1155
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods for enhancing the therapeutic and adjuvant use of ***IL*** - ***12*** by reducing unwanted transient immunosuppression caused by ***IL*** - ***12*** or by high doses thereof involve co-administering ***IL*** - ***12*** with an effective amount of an agent that inhibits or neutralizes nitric oxide (NO) in vivo. Enhanced vaccine therapy involves co-administering the ***IL*** - ***12*** adjuvant, a selected vaccine antigen and the NO inhibiting/neutralizing agent. Additionally, the toxicity of ***IL*** - ***12*** treatment may be reduced by co-administering ***IL*** - ***12*** with an effective amount of the NO inhibiting or neutralizing agent. A therapeutic composition characterized by reduced toxicity in mammals contains ***IL*** - ***12*** , preferably a low dose thereof, and an NO inhibiting or neutralizing agent in a pharmaceutically acceptable carrier. A vaccine composition contains an effective adjuvanting amount of ***IL*** - ***12*** , an effective amount of an NO inhibiting or neutralizing agent, and an effective protective amount of a vaccine antigen in a pharmaceutically acceptable carrier.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d 1- ibib abs
YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y

L3 ANSWER 1 OF 3 USPATFULL on STN
ACCESSION NUMBER: 2002:156684 USPATFULL
TITLE: Methods and compositions for enhancing the immunostimulatory effect of ***interleukin*** - ***12***
INVENTOR(S): Trinchieri, Giorgio, Charly, FRANCE
Lee, William M. F., Wynnewood, PA, UNITED STATES
Koblish, Holly, Yardley, PA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002081277	A1	20020627
APPLICATION INFO.:	US 2002-79068	A1	20020220 (10)

RELATED APPLN. INFO.: Division of Ser. No. US 1999-395038, filed on 13 Sep 1999, GRANTED, Pat. No. US 6375944

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-101698P	19980925 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HOWSON AND HOWSON, ONE SPRING HOUSE CORPORATION CENTER, BOX 457, 321 NORRISTOWN ROAD, SPRING HOUSE, PA, 19477
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 7 Drawing Page(s)
LINE COUNT: 1155
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods for enhancing the therapeutic and adjuvant use of ***IL*** - ***12*** by reducing unwanted transient immunosuppression caused by ***IL*** - ***12*** or by high doses thereof involve co-administering ***IL*** - ***12*** with an effective amount of an agent that inhibits or neutralizes nitric oxide (NO) in vivo. Enhanced vaccine therapy involves co-administering the ***IL*** - ***12*** adjuvant, a selected vaccine antigen and the NO inhibiting/neutralizing agent. Additionally, the toxicity of ***IL*** - ***12*** treatment may be reduced by co-administering ***IL*** - ***12*** with an effective amount of the NO inhibiting or neutralizing agent. A therapeutic composition characterized by reduced toxicity in mammals contains ***IL*** - ***12*** , preferably a low dose thereof, and an NO inhibiting or neutralizing agent in a pharmaceutically acceptable carrier. A vaccine composition contains an effective adjuvanting amount of ***IL*** - ***12*** , an effective amount of an NO inhibiting or neutralizing agent, and an effective protective amount of a vaccine antigen in a pharmaceutically acceptable carrier.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 2 OF 3 USPATFULL on STN
ACCESSION NUMBER: 2002:87988 USPATFULL
TITLE: Methods and compositions for enhancing the immunostimulatory effect of ***interleukin*** - ***12***
INVENTOR(S): Trinchieri, Giorgio, Charly, FRANCE
Lee, William M. F., Wynnewood, PA, United States
Koblish, Holly, Yardley, PA, United States
PATENT ASSIGNEE(S): The Wistar Institute of Anatomy and Biology, Philadelphia, PA, United States (U.S. corporation)
The Trustees of the University of Pennsylvania, Philadelphia, PA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6375944	B1	20020423
APPLICATION INFO.:	US 1999-395038		19990913 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-101698P	19980925 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Mertz, Prema
ASSISTANT EXAMINER: Prasad, Sarada C
LEGAL REPRESENTATIVE: Howson and Howson
NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Figure(s); 7 Drawing Page(s)
LINE COUNT: 1207
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods for enhancing the therapeutic and adjuvant use of ***IL*** - ***12*** by reducing unwanted transient immunosuppression caused by ***IL*** - ***12*** or by high doses thereof involve co-administering ***IL*** - ***12*** with an effective amount of an agent that inhibits or neutralizes nitric oxide (NO) in vivo. Enhanced vaccine therapy involves co-administering the ***IL*** - ***12*** adjuvant, a selected vaccine antigen and the NO inhibiting/neutralizing agent. Additionally, the toxicity of ***IL*** - ***12*** treatment may be reduced by co-administering ***IL*** - ***12*** with an effective amount of the NO inhibiting or neutralizing agent. A therapeutic composition characterized by reduced toxicity in mammals contains ***IL*** - ***12*** , preferably a low dose thereof, and an NO inhibiting or neutralizing agent in a pharmaceutically acceptable carrier. A vaccine composition contains an effective adjuvanting amount of ***IL*** - ***12*** , an effective amount of an NO inhibiting or neutralizing agent, and an effective protective amount of a vaccine antigen in a pharmaceutically acceptable carrier.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2000:60896 CAPLUS
TITLE: Papers to Appear in Forthcoming Issues
AUTHOR(S): Anon.
SOURCE: Cellular Immunology (1999), 198(2), 144
CODEN: CLIMB8; ISSN: 0008-8749
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal; Miscellaneous
LANGUAGE: English
AB Human Leptin Enhances Activation and Proliferation of Human Circulating T
Lymphocytes. Consuelo Martin-Romero, Jose Santos-Alvarez, Raimundo
Goberna, and Victor Sanchez-Margalet. (Received 6/17/99; accepted
11/24/99.)Induction of ***Interleukin*** - ***12*** /p40 by
Superantigens in Macrophages Is Mediated by Activation of Nuclear
Factor-.kappa.B. Caigan Du and Subramaniam Sriram. (Received 9/16/99;
accepted 11/29/99.).alpha.6.beta.1 Integrin (VLA-6) Mediates Leukocyte
Tether and Arrest on Laminin under Physiol. Shear Flow. Joji Kitayama,
Shigeo Ikeda, Kyoko Kumagai, Hideaki Saito, and Hirokazu Nagawa. (Received
8/26/99; accepted 12/1/99.)Macrophage-Derived ***Nitric***
Oxide ***Inhibits*** the Proliferation of Activated T Helper
Cells and Is Induced during Antigenic Stimulation of Resting T Cells.
Roel C. van der Veen, Therese A. Dietlin, J. Dixon Gray, and Wendy
Gilmore. (Received 8/26/99; accepted 12/1/99.)Synthetic Melanin Suppresses
Prodn. of Proinflammatory Cytokines. Nahid Mohagheghpour, Nahid Waleh,
Stephen J. Garger, Linda Dousman, Laurence K. Grill, and Daniel Tuse.
(Received 7/12/99; accepted 12/6/99.)Cell-Specific Inhibition of Inducible
Nitric Oxide Synthase Activation by Leflunomide. V. Jankovic, T.
Samardzic, S. Stosic-Grujicic, D. Popadic, and V. Trajkovic. (Received
7/13/99; accepted 12/6/99.)Regulation of Human Natural Killer Cell
Migration and Proliferation by the Exodus Subfamily of CC Chemokines.
Michael J. Robertson, Brian T. Williams, Kent Christopherson II, Zacharie
Brahmi, and Robert Hromas. (Received 4/27/99; accepted
12/7/99.).alpha.-Galactosylceramide Induces Early B-Cell Activation
through IL-4 Prodn. by NKT Cells. Hidemitsu Kitamura, Akio Ohta, Masashi
Sekimoto, Marimo Sato, Kenji Iwakabe, Minoru Nakui, Takashi Yahata, Hongxu
Meng, Toshiaki Koda, Shin-ichiro Nishimura, Tetsu Kawano, Masaru
Taniguchi, and Takashi Nishimura. (Received 7/26/99; accepted 12/9/99.).
(c) 1999 Academic Press.

=> s (Il-12 or interleukin-12) and (NO (w) inhibit? or NO (w) neutraliz?)
5 FILES SEARCHED...
L4 164 (IL-12 OR INTERLEUKIN-12) AND (NO (W) INHIBIT? OR NO (W) NEUTRAL IZ?)

=> duplicate remove l4
DUPLICATE PREFERENCE IS 'CAPLUS, MEDLINE, BIOSIS, EMBASE, USPATFULL, BIOTECHNO'
KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n
PROCESSING COMPLETED FOR L4
L5 74 DUPLICATE REMOVE L4 (90 DUPLICATES REMOVED)

=> d 1- ibib abs
YOU HAVE REQUESTED DATA FROM 74 ANSWERS - CONTINUE? Y/(N):y

L5 ANSWER 1 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2005:10995 USPATFULL
TITLE: Methods of screening for a candidate modulator of
glucokinase
INVENTOR(S): Rizzo, Mark A., Nashville, TN, UNITED STATES
Piston, David W., Nashville, TN, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005009129	A1	20050113
APPLICATION INFO.:	US 2004-838167	A1	20040503 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-467885P	20030505 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Steven L. Highlander, FULBRIGHT & JAWORSKI L.L.P., 600
Congress Avenue, Suite 2400, Austin, TX, 78701
NUMBER OF CLAIMS: 49
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Page(s)
LINE COUNT: 1679

AB The present invention relates to providing novel therapeutics for
treating diabetes other glycemic disorders. Such therapeutics involve
the signaling pathways that contribute to regulation of
glucose-stimulated insulin secretion. Of particular interest are
modulators of a key component in the glucokinase pathway. Thus, the
present provides methods of screening for modulators of glucokinase
activity, expression, translocation, conformation, nitrosylation and
interaction with other molecules as useful target for pharmacological
manipulation in the treatment of diabetes and other glycemic disorders.

L5 ANSWER 2 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2004:326849
USPATFULL
TITLE: Method for regulating the expression of genes carried
on a viral vector
INVENTOR(S): Inoue, Makoto, Ibaraki, JAPAN
Iida, Akihiro, Ibaraki, JAPAN
Hasegawa, Mamoru, Ibaraki, JAPAN

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004258668	A1	20041223
APPLICATION INFO.:	US 2004-489394	A1	20040813 (10)
	WO 2002-JP10065		20020927

	NUMBER	DATE
PRIORITY INFORMATION:	JP 2001-298223	20010927

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: CLARK & ELBING LLP, 101 FEDERAL STREET, BOSTON, MA, 02110
NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 13 Drawing Page(s)
LINE COUNT: 810
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present inventors revealed that non-steroidal anti-inflammatory
drugs and muscle relaxants suppressed the expression of genes carried on
viral vectors. These agents also suppressed viral vector cytotoxicity.
The present invention provides a method for regulating the expression of
genes carried on viruses by using non-steroidal anti-inflammatory drugs
and/or muscle relaxants. The effect of these agents is reversible, and
viral vector gene expression and cytotoxicity increased after
termination of agent administration. The agents of the present invention
are useful for regulating the expression of viral and therapeutic genes,
and for suppressing viral vector cytotoxicity in gene therapy using
viral vectors.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 3 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2004:313900
USPATFULL
TITLE: Auditory nerve protection and re-growth
INVENTOR(S): Miller, Josef M., Ann Arbor, MI, UNITED STATES
Altschuler, Richard A., Ann Arbor, MI, UNITED STATES
Raphael, Yehoash, Ann Arbor, MI, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004247570	A1	20041209
APPLICATION INFO.:	US 2003-345731	A1	20030116 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-349799P	20020117 (60)
	US 2002-351870P	20020125 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Tanya A Arenson, MEDLEN & CARROLL LLP, Suite 350, 101 Howard Street, San Francisco, CA, 94105
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 21 Drawing Page(s)
LINE COUNT: 2235
AB The present invention relates to compositions and methods for the
protection and restoration of hearing. In particular, the present
invention relates to treatments to facilitate the protection and
re-growth of the auditory nerve. The present invention further provides
methods of preventing hair cell loss and the accompanying loss in

hearing. The present invention thus provides novel interventions for a
variety of hearing impairments.

L5 ANSWER 4 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2004:306481 USPATFULL
TITLE: Use of unmethylatd cpg
INVENTOR(S): De Simone, Claudio, Ardea Rm, ITALY

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004241149	A1	20041202
APPLICATION INFO.:	US 2004-488606	A1	20040303 (10)
	WO 2002-IT534		20020809

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-316953P	20010905 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: NIXON & VANDERHYE, PC, 1100 N GLEBE ROAD, 8TH FLOOR,
ARLINGTON, VA, 22201-4714
NUMBER OF CLAIMS: 28
EXEMPLARY CLAIM: 1
LINE COUNT: 897
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Lactic acid bacteria containing unmethylated cytosine-guanine (CpG)
dinucleotide are used to positively affect the immune response in a
subject having or at risk of having an inflammatory response to
lipopolysaccharides.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 5 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2004:281091 USPATFULL
TITLE: Compositions for, and methods of, treating
atherosclerosis
INVENTOR(S): Romanczyk,, Leo J., JR., Hackettstown, NJ, UNITED STATES
Schmitz, Harold H., Branchburg, NJ, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004220392	A1	20041104
APPLICATION INFO.:	US 2004-770969	A1	20040506 (10)
RELATED APPLN. INFO.:	Division of Ser. No. US 2002-127817, filed on 22 Apr 2002, PENDING Continuation of Ser. No. US 2001-776649, filed on 5 Feb 2001, GRANTED, Pat. No. US 6638971 Continuation of Ser. No. US 1997-831245, filed on 2 Apr 1997, GRANTED, Pat. No. US 6297273 Continuation-in-part of Ser. No. US 1996-631661, filed on 2 Apr 1996, ABANDONED Continuation of Ser. No. US 2000-717893, filed on 21 Nov 2000, GRANTED, Pat. No. US 6670390 Continuation of Ser. No. US 1997-831245, filed on 2 Apr 1997, GRANTED, Pat. No. US 6297273 Continuation-in-part of Ser. No. US 1996-631661, filed on 2 Apr 1996, ABANDONED		

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: NADA JAIN, P.C., 560 White Plains Road, Suite 460,
Tarrytown, NY, 10591
NUMBER OF CLAIMS: 17
EXEMPLARY CLAIM: CLM-1-208
NUMBER OF DRAWINGS: 242 Drawing Page(s)
LINE COUNT: 4732
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed and claimed are cocoa extracts, compounds, combinations
thereof and compositions containing the same, such as polyphenols or
procyanidins, methods for preparing such extracts, compounds and
compositions, as well as uses for them, especially a polymeric compound
of the formula A.sub.n, wherein A is a monomer of the formula:
##STR1##

wherein n is an integer from 2 to 18, such that there is at least one
terminal monomeric unit A, and one or a plurality of additional
monomeric units;

R is 3-(.alpha.)-OH, 3-(.beta.)-OH, 3-(.alpha.)-o-sugar, or
3-(.beta.)-O-sugar;

bonding between adjacent monomers takes place at positions 4, 6 or 8;

a bond of an additional monomeric unit in position 4 has alpha or beta
stereochemistry;

X, Y and Z are selected from the group consisting of monomeric unit A,
hydrogen, and a sugar, with the provisos that as to the at least one
terminal monomeric unit, bonding of the additional monomeric unit
thereto (the bonding of the additional monomeric unit adjacent to the
terminal monomeric unit) is at position 4 and optionally Y=Z=hydrogen;

the sugar is optionally substituted with a phenolic moiety, at any
position on the sugar, for instance via an ester bond, and

pharmaceutically acceptable salts or derivatives thereof (including
oxidation products).

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 6 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2004:69535 USPATFULL
TITLE: Stat3 agonists and antagonists and therapeutic uses
thereof
INVENTOR(S): Yu, Hua, Tampa, FL, UNITED STATES
Pardoll, Drew, Brookeville, MD, UNITED STATES
Jove, Richard, Tampa, FL, UNITED STATES
Dalton, William, Tampa, FL, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004052762	A1	20040318
APPLICATION INFO.:	US 2003-380020	A1	20030902 (10)
	WO 2001-US28254		20010910

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: JONES DAY, 222 EAST 41ST STREET, NEW YORK, NY, 10017
NUMBER OF CLAIMS: 49
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 13 Drawing Page(s)
LINE COUNT: 3467
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to methods for modulating, i.e., agonizing
or antagonizing, Stat3 (Signal Transducer and Activator of
Transcription3) signaling activity for use in gene therapy. Inhibition
and/or activation of Stat3 signaling is an effective approach to
modulate angiogenesis and the immune response for treatment and/or
prevention of inflammation, infection, inflammation, immune disorders,
and ischemia.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 7 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2004:57395 USPATFULL
TITLE: IL-17 homologous polypeptides and therapeutic uses
thereof
INVENTOR(S): Chen, Jian, Princeton, NJ, UNITED STATES
Filvaroff, Ellen, San Francisco, CA, UNITED STATES
Fong, Sherman, Alameda, CA, UNITED STATES
Goddard, Audrey, San Francisco, CA, UNITED STATES
Godowski, Paul, Hillsborough, CA, UNITED STATES
Grimaldi, Christopher, San Francisco, CA, UNITED STATES
Gurney, Austin, Belmont, CA, UNITED STATES
Li, Hanzhong, San Mateo, CA, UNITED STATES
Hillan, Kenneth, San Francisco, CA, UNITED STATES
Tumas, Daniel, Orinda, CA, UNITED STATES
VanLookeren, Menno, San Francisco, CA, UNITED STATES
Vandlen, Richard, Hillsborough, CA, UNITED STATES
Watanabe, Colin K., Moraga, CA, UNITED STATES
Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES
Wood, William I., Hillsborough, CA, UNITED STATES
Yansura, Daniel, Pacifica, CA, UNITED STATES
PATENT ASSIGNEE(S): Genentech, Inc., South San Francisco, CA (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004043397	A1	20040304

APPLICATION INFO.: US 2003-408385 A1 20030407 (10)
RELATED APPLN. INFO.: Division of Ser. No. US 2000-747259, filed on 20 Dec
2000, GRANTED, Pat. No. US 6569645 Continuation-in-part
of Ser. No. WO 2000-US30873, filed on 10 Nov 2000,
PENDING Continuation-in-part of Ser. No. WO
2000-US23328, filed on 24 Aug 2000, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-175481P	20000111 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: GENENTECH, INC., 1 DNA WAY, SOUTH SAN FRANCISCO, CA, 94080
NUMBER OF CLAIMS: 60
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 46 Drawing Page(s)
LINE COUNT: 8591
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention is directed to novel polypeptides and to nucleic
acid molecules encoding those polypeptides. Also provided herein are
vectors and host cells comprising those nucleic acid sequences, chimeric
polypeptide molecules comprising the polypeptides of the present
invention fused to heterologous polypeptide sequences, antibodies which
bind to the polypeptides of the present invention and to methods for
producing the polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 8 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2004:46811 USPATFULL
TITLE: Procyanidin and cyclo-oxygenase modulator compositions
INVENTOR(S): Romanczyk, Jr., Leo J., Hackettstown, NJ, United States
Schmitz, Harold H., Branchburg, NJ, United States
PATENT ASSIGNEE(S): Mars, Incorporated, McLean, VA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6696485	B1	20040224
APPLICATION INFO.:	US 2002-268718		20021010 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2000-717893, filed on 21
Nov 2000 Continuation of Ser. No. US 2001-776649, filed
on 5 Feb 2001 Continuation of Ser. No. US 2002-127817,
filed on 22 Apr 2002
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Solola, Taofiq
LEGAL REPRESENTATIVE: Nada Jain, P.C., Jain, Nada
NUMBER OF CLAIMS: 14
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 54 Drawing Figure(s); 241 Drawing Page(s)
LINE COUNT: 4397
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention relates to compositions comprising a cyclo-oxygenase
modulator in combination with cocoa procyanidin monomers and/or
oligomers, wherein the cyclo-oxygenase modulator is a non-steroidal
anti-inflammatory drug such as aspirin. Such compositions may be used
for the treatment of cardiovascular related disorders.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 9 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2004:4504 USPATFULL
TITLE: Tumor necrosis factor receptor 2
INVENTOR(S): Stanton, Jr., Vincent P., Belmont, MA, United States
PATENT ASSIGNEE(S): Nuvelo, Inc., Sunnyvale, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6673908	B1	20040106
APPLICATION INFO.:	US 2001-968455		20011001 (9)

RELATED APPLN. INFO.: Division of Ser. No. US 2000-649035, filed on 25 Aug
2000 Continuation-in-part of Ser. No. US 2000-590749,
filed on 8 Jun 2000, now abandoned Continuation-in-part
of Ser. No. US 2000-495780, filed on 1 Feb 2000, now

abandoned Continuation-in-part of Ser. No. US
2000-492712, filed on 27 Jan 2000, now abandoned
Continuation-in-part of Ser. No. WO 2000-US1392, filed
on 20 Jan 2000 Continuation-in-part of Ser. No. US
968455 Continuation-in-part of Ser. No. US 1999-451252,
filed on 29 Nov 1999, now abandoned
Continuation-in-part of Ser. No. US 1999-427835, filed
on 26 Oct 1999, now abandoned Continuation-in-part of
Ser. No. US 1999-414330, filed on 6 Oct 1999, now
abandoned Continuation-in-part of Ser. No. US
1999-389993, filed on 3 Sep 1999, now abandoned
Continuation-in-part of Ser. No. US 1999-370841, filed
on 9 Aug 1999, now abandoned Continuation-in-part of
Ser. No. US 1999-300747, filed on 26 Apr 1999, now
abandoned

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-131334P	19990426 (60)
	US 1999-131191P	19990426 (60)
	US 1999-121047P	19990222 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Benzion, Gary
ASSISTANT EXAMINER: Chakrabarti, Arun Kr.
LEGAL REPRESENTATIVE: Fish & Richardson P.C.
NUMBER OF CLAIMS: 10
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 0 Drawing Figure(s); 0 Drawing Page(s)
LINE COUNT: 17463
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present disclosure describes the use of genetic variance information
for genes involved in inflammatory or immunologic disease, disorder, or
dysfunction. The variance information is indicative of the expected
response of a patient to a method of treatment. Methods of determining
relevant variance information and additional methods of using such
variance information are also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 10 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2004:194249 CAPLUS
DOCUMENT NUMBER: 140:269493
TITLE: Inhibition of ***Interleukin*** - ***12*** p40
Transcription and NF-.kappa.B Activation by Nitric
Oxide in Murine Macrophages and Dendritic Cells
AUTHOR(S): Xiong, Huabao; Zhu, Chen; Li, Fengling; Hegazi,
Refaat; He, Kaili; Babyatsky, Mark; Bauer, Anthony J.;
Plevy, Scott E.
CORPORATE SOURCE: Immunobiology Center, The Mount Sinai School of
Medicine, New York, NY, 10029, USA
SOURCE: Journal of Biological Chemistry (2004), 279(11),
10776-10783
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular
Biology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Nitric oxide (NO), an important effector mol. of the innate immune system,
can also regulate adaptive immunity. In this study, the mol. effects of
NO on the Toll-like receptor signaling pathway were detd. using
interleukin - ***12*** (***IL*** - ***12***) as an
immunol. relevant target gene. The principal conclusion of these expts.
is that ***NO*** ***inhibits*** IL-1 receptor-assocd. kinase
(IRAK) activity and attenuates the mol. interaction between tumor necrosis
factor receptor-assocd. factor-6 and IRAK. As a consequence, the NO donor
S-nitroso-N-acetylpenicillamine (SNAP) inhibits lipopolysaccharide
(LPS)-induced ***IL*** - ***12*** p40 mRNA expression, protein
prodn., and promoter activity in murine macrophages, dendritic cells, and
the murine macrophage cell line RAW 264.7. Splenocytes from inducible
nitric-oxide synthase-deficient mice demonstrate markedly increased
IL - ***12*** p40 protein and mRNA expression compared with wild
type splenocytes. The inhibitory action of NO on ***IL*** - ***12***
p40 is independent of the cytokine IL-10. The effects of NO can be

directly attributed to inhibition of NF-.kappa.B activation through
IRAK-dependent pathways. Accordingly, SNAP strongly reduces LPS-induced
NF-.kappa.B DNA binding to the p40 promoter and inhibits LPS-induced
I.kappa.B phosphorylation. Similarly, NO attenuates IL-1.beta.-induced
NF-.kappa.B activation. These expts. provide another example of how an
innate immune mol. may have a profound effect on adaptive immunity.
REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 11 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:299854
USPATFULL
TITLE: Combined compositions for tumor vasculature coagulation and treatment
INVENTOR(S): Thorpe, Philip E., Dallas, TX, UNITED STATES
King, Steven W., Rancho Santa Margarita, CA, UNITED STATES
Gottstein, Claudia, Dallas, TX, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003211075	A1	20031113
APPLICATION INFO.:	US 2002-259244	A1	20020927 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-325532P	20010927 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Shelley P.M. Fussey, Ph.D., Williams, Morgan & Amerson, P.C., Suite 1100, 10333 Richmond Avenue, Houston, TX, 77042
NUMBER OF CLAIMS: 43
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 9999
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are various defined combinations of agents for use in improved
anti-vascular therapies and coagulative tumor treatment. Particularly
provided are combined treatment methods, and associated compositions,
pharmaceuticals, medicaments, kits and uses, which together function
surprisingly effectively in the treatment of vascularized tumors. The
invention preferably involves a component or treatment step that
enhances the effectiveness of therapy using targeted or non-targeted
coagulants to cause tumor vasculature thrombosis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 12 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:288693
USPATFULL
TITLE: IL-17 homologous polypeptides and therapeutic uses thereof
INVENTOR(S): Chen, Jian, Princeton, NJ, UNITED STATES
Filvaroff, Ellen, San Francisco, CA, UNITED STATES
Fong, Sherman, Alameda, CA, UNITED STATES
Goddard, Audrey, San Francisco, CA, UNITED STATES
Godowski, Paul, Burlingame, CA, UNITED STATES
Grimaldi, J. Christopher, San Francisco, CA, UNITED STATES
Gurney, Austin, Belmont, CA, UNITED STATES
Li, Hanzhong, San Mateo, CA, UNITED STATES
Hillan, Kenneth, San Francisco, CA, UNITED STATES
Hymowitz, Sarah G., San Francisco, CA, UNITED STATES
Tumas, Daniel, Orinda, CA, UNITED STATES
Starovasnik, Melissa A., San Francisco, CA, UNITED STATES
VanLookeren, Menno, San Francisco, CA, UNITED STATES
Vandlen, Richard, Hillsborough, CA, UNITED STATES
Watanabe, Colin, Moraga, CA, UNITED STATES
Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES
Wood, William I., Hillsborough, CA, UNITED STATES
Yansura, Daniel, Pacifica, CA, UNITED STATES
PATENT ASSIGNEE(S): Genentech, Inc., South San Francisco, CA, UNITED STATES, 94080 (U.S. corporation)

	NUMBER	KIND	DATE

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PATENT INFORMATION: US 2003203451 A1 20031030
APPLICATION INFO.: US 2003-458442 A1 20030610 (10)
RELATED APPLN. INFO.: Division of Ser. No. US 2001-874503, filed on 5 Jun
2001, PENDING Continuation-in-part of Ser. No. US
2001-816744, filed on 22 Mar 2001, GRANTED, Pat. No. US
6579520 Continuation-in-part of Ser. No. WO
2001-US6520, filed on 28 Feb 2001, PENDING
Continuation-in-part of Ser. No. US 2000-747259, filed
on 20 Dec 2000, GRANTED, Pat. No. US 6569645
Continuation-in-part of Ser. No. WO 2000-US23328, filed
on 24 Aug 2000, PENDING
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: GENENTECH, INC., 1 DNA WAY, SOUTH SAN FRANCISCO, CA,
94080
NUMBER OF CLAIMS: 60
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 55 Drawing Page(s)
LINE COUNT: 8852
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention is directed to novel polypeptides having sequence
identity with IL-17, IL-17 receptors and to nucleic acid molecules
encoding those polypeptides. Also provided herein are vectors and host
cells comprising those nucleic acid sequences, chimeric polypeptide
molecules comprising the polypeptides of the present invention fused to
heterologous polypeptide sequences, antibodies which bind to the
polypeptides of the present invention and to methods for producing the
polypeptides of the present invention. Further provided herein are
methods for treating degenerative cartilaginous disorders and other
inflammatory diseases.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 13 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:282701 USPATFULL
TITLE: IL-17 homologous polypeptides and therapeutic uses
thereof
INVENTOR(S): Chen, Jian, Princeton, NJ, UNITED STATES
Filvaroff, Ellen, San Francisco, CA, UNITED STATES
Fong, Sherman, Alameda, CA, UNITED STATES
Goddard, Audrey, San Francisco, CA, UNITED STATES
Godowski, Paul, Hillsborough, CA, UNITED STATES
Grimaldi, Christopher, San Francisco, CA, UNITED STATES
Gurney, Austin, Belmont, CA, UNITED STATES
Li, Hanzhong, San Mateo, CA, UNITED STATES
Hillan, Kenneth, San Francisco, CA, UNITED STATES
Tumas, Daniel, Orinda, CA, UNITED STATES
VanLookeren, Menno, San Francisco, CA, UNITED STATES
Vandlen, Richard, Hillsborough, CA, UNITED STATES
Watanabe, Colin, Moraga, CA, UNITED STATES
Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES
Wood, William I., Hillsborough, CA, UNITED STATES
Yansura, Daniel, Pacifica, CA, UNITED STATES
PATENT ASSIGNEE(S): Genentech, Inc., South San Francisco, CA, UNITED STATES
(U.S. corporation)

NUMBER KIND DATE
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PATENT INFORMATION: US 2003199044 A1 20031023
APPLICATION INFO.: US 2003-410552 A1 20030408 (10)
RELATED APPLN. INFO.: Division of Ser. No. US 2000-747259, filed on 20 Dec
2000, GRANTED, Pat. No. US 6569645 Continuation-in-part
of Ser. No. WO 2000-US23328, filed on 24 Aug 2000,
PENDING
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: GENENTECH, INC., 1 DNA WAY, SOUTH SAN FRANCISCO, CA,
94080
NUMBER OF CLAIMS: 60
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 47 Drawing Page(s)
LINE COUNT: 8602
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention is directed to novel polypeptides and to nucleic

acid molecules encoding those polypeptides. Also provided herein are
vectors and host cells comprising those nucleic acid sequences, chimeric
polypeptide molecules comprising the polypeptides of the present
invention fused to heterologous polypeptide sequences, antibodies which
bind to the polypeptides of the present invention and to methods for
producing the polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 14 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:277229
USPATFULL
TITLE: Inhibitors of nitric oxide synthase
INVENTOR(S): Singh, Inderjit, Mount Pleasant, SC, UNITED STATES
PATENT ASSIGNEE(S): MUSC Foundation for Research Development (U.S.
corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003195256	A1	20031016
APPLICATION INFO.:	US 2002-273557	A1	20021018 (10)

RELATED APPLN. INFO.: Division of Ser. No. US 2000-579791, filed on 25 May
2000, GRANTED, Pat. No. US 6511800 Continuation of Ser.
No. WO 1998-US25360, filed on 25 Nov 1998, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 1997-66839P	19971125 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Michael R. Krawzsenek, FULBRIGHT & JAWORSKI L.L.P.,
Suite 2400, 600 Congress Avenue, Austin, TX, 78701
NUMBER OF CLAIMS: 75
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Page(s)
LINE COUNT: 7728
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The current invention discloses novel methods for the inhibition of
inducible nitric oxide synthesis (iNOS) and the production of NO.
Methods of inhibiting the induction of proinflammatory cytokines are
also described. Methods of treating various disease states, such as
X-linked adrenoleukodystrophy, multiple sclerosis, Alzheimer's and
septic shock using inhibitors of iNOS and cytokine induction are
disclosed. The inhibitors include the exemplary compounds lovastatin, a
sodium salt of phenylacetic acid (NaPA), FPT inhibitor II, N-acetyl
cysteine (NAC), and cAMP.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 15 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:265291
USPATFULL
TITLE: IL-17 homologous polypeptides and therapeutic uses
thereof
INVENTOR(S): Chen, Jian, Princeton, NJ, UNITED STATES
Filvaroff, Ellen, San Francisco, CA, UNITED STATES
Fong, Sherman, Alameda, CA, UNITED STATES
Goddard, Audrey, San Francisco, CA, UNITED STATES
Godowski, Paul J., Hillsborough, CA, UNITED STATES
Grimaldi, Christopher, San Francisco, CA, UNITED STATES
Gurney, Austin, Belmont, CA, UNITED STATES
Li, Hanzhong, San Mateo, CA, UNITED STATES
Hillan, Kenneth, San Francisco, CA, UNITED STATES
Tumas, Daniel, Orinda, CA, UNITED STATES
VanLookeren, Menno, San Francisco, CA, UNITED STATES
Vandlen, Richard, Hillsborough, CA, UNITED STATES
Watanabe, Colin, Moraga, CA, UNITED STATES
Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES
Wood, William I., Hillsborough, CA, UNITED STATES
Yansura, Daniel, Pacifica, CA, UNITED STATES
PATENT ASSIGNEE(S): Genentech, Inc., South San Francisco, CA, UNITED STATES
(U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003186306	A1	20031002

APPLICATION INFO.: US 2003-410374 A1 20030408 (10)
RELATED APPLN. INFO.: Division of Ser. No. US 2000-747259, filed on 20 Dec
2000, GRANTED, Pat. No. US 6569645 Continuation-in-part
of Ser. No. WO 2000-US23328, filed on 24 Aug 2000,
PENDING
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: GENENTECH, INC., 1 DNA WAY, SOUTH SAN FRANCISCO, CA,
94080
NUMBER OF CLAIMS: 60
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 47 Drawing Page(s)
LINE COUNT: 8095
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention is directed to novel polypeptides and to nucleic
acid molecules encoding those polypeptides. Also provided herein are
vectors and host cells comprising those nucleic acid sequences, chimeric
polypeptide molecules comprising the polypeptides of the present
invention fused to heterologous polypeptide sequences, antibodies which
bind to the polypeptides of the present invention and to methods for
producing the polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 16 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:258639 USPATFULL
TITLE: 207 human secreted proteins
INVENTOR(S): Ni, Jian, Germantown, MD, UNITED STATES
Ebner, Reinhard, Gaithersburg, MD, UNITED STATES
LaFleur, David W., Washington, DC, UNITED STATES
Moore, Paul A., Germantown, MD, UNITED STATES
Olsen, Henrik S., Gaithersburg, MD, UNITED STATES
Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
Soppet, Daniel R., Centreville, VA, UNITED STATES
Young, Paul E., Gaithersburg, MD, UNITED STATES
Shi, Yanggu, Gaithersburg, MD, UNITED STATES
Florence, Kimberly A., Rockville, MD, UNITED STATES
Wei, Ying-Fei, Berkeley, CA, UNITED STATES
Florence, Charles, Rockville, MD, UNITED STATES
Hu, Jing-Shan, Mountain View, CA, UNITED STATES
Li, Yi, Sunnyvale, CA, UNITED STATES
Kyaw, Hla, Frederick, MD, UNITED STATES
Fischer, Carrie L., Burke, VA, UNITED STATES
Ferrie, Ann M., Painted Post, NY, UNITED STATES
Fan, Ping, Potomac, MD, UNITED STATES
Feng, Ping, Gaithersburg, MD, UNITED STATES
Endress, Gregory A., Florence, MA, UNITED STATES
Dillon, Patrick J., Carlsbad, CA, UNITED STATES
Carter, Kenneth C., North Potomac, MD, UNITED STATES
Brewer, Laurie A., St. Paul, MN, UNITED STATES
Yu, Guo-Liang, Berkeley, CA, UNITED STATES
Zeng, Zhizhen, Lansdale, PA, UNITED STATES
Greene, John M., Gaithersburg, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003181692	A1	20030925
APPLICATION INFO.:	US 2001-933767	A1	20010822 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. WO 2001-US5614, filed
on 21 Feb 2001, PENDING
Continuation-in-part of Ser.
No. US 1998-205258, filed on 4 Dec 1998, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-184836P	20000224 (60)
	US 2000-193170P	20000329 (60)
	US 1997-48885P	19970606 (60)
	US 1997-49375P	19970606 (60)
	US 1997-48881P	19970606 (60)
	US 1997-48880P	19970606 (60)
	US 1997-48896P	19970606 (60)
	US 1997-49020P	19970606 (60)
	US 1997-48876P	19970606 (60)
	US 1997-48895P	19970606 (60)
	US 1997-48884P	19970606 (60)

US 1997-48894P 19970606 (60)
US 1997-48971P 19970606 (60)
US 1997-48964P 19970606 (60)
US 1997-48882P 19970606 (60)
US 1997-48899P 19970606 (60)
US 1997-48893P 19970606 (60)
US 1997-48900P 19970606 (60)
US 1997-48901P 19970606 (60)
US 1997-48892P 19970606 (60)
US 1997-48915P 19970606 (60)
US 1997-49019P 19970606 (60)
US 1997-48970P 19970606 (60)
US 1997-48972P 19970606 (60)
US 1997-48916P 19970606 (60)
US 1997-49373P 19970606 (60)
US 1997-48875P 19970606 (60)
US 1997-49374P 19970606 (60)
US 1997-48917P 19970606 (60)
US 1997-48949P 19970606 (60)
US 1997-48974P 19970606 (60)
US 1997-48883P 19970606 (60)
US 1997-48897P 19970606 (60)
US 1997-48898P 19970606 (60)
US 1997-48962P 19970606 (60)
US 1997-48963P 19970606 (60)
US 1997-48877P 19970606 (60)
US 1997-48878P 19970606 (60)
US 1997-57645P 19970905 (60)
US 1997-57642P 19970905 (60)
US 1997-57668P 19970905 (60)
US 1997-57635P 19970905 (60)
US 1997-57627P 19970905 (60)
US 1997-57667P 19970905 (60)
US 1997-57666P 19970905 (60)
US 1997-57764P 19970905 (60)
US 1997-57643P 19970905 (60)
US 1997-57769P 19970905 (60)
US 1997-57763P 19970905 (60)
US 1997-57650P 19970905 (60)
US 1997-57584P 19970905 (60)
US 1997-57647P 19970905 (60)
US 1997-57661P 19970905 (60)
US 1997-57662P 19970905 (60)
US 1997-57646P 19970905 (60)
US 1997-57654P 19970905 (60)
US 1997-57651P 19970905 (60)
US 1997-57644P 19970905 (60)
US 1997-57765P 19970905 (60)
US 1997-57762P 19970905 (60)
US 1997-57775P 19970905 (60)
US 1997-57648P 19970905 (60)
US 1997-57774P 19970905 (60)
US 1997-57649P 19970905 (60)
US 1997-57770P 19970905 (60)
US 1997-57771P 19970905 (60)
US 1997-57761P 19970905 (60)
US 1997-57760P 19970905 (60)
US 1997-57776P 19970905 (60)
US 1997-57778P 19970905 (60)
US 1997-57629P 19970905 (60)
US 1997-57628P 19970905 (60)
US 1997-57777P 19970905 (60)
US 1997-57634P 19970905 (60)
US 1997-70923P 19971218 (60)
US 1998-92921P 19980715 (60)
US 1998-94657P 19980730 (60)
US 1997-70923P 19971218 (60)
US 1998-92921P 19980715 (60)
US 1998-94657P 19980730 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Page(s)
LINE COUNT: 32746
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel human secreted proteins and isolated nucleic acids containing the coding regions of the genes encoding such proteins. Also provided are vectors, host cells, antibodies, and recombinant methods for producing human secreted proteins. The invention further relates to diagnostic and therapeutic methods useful for diagnosing and treating diseases, disorders, and/or conditions related to these novel human secreted proteins.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 17 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:257205 USPATFULL
TITLE: IL-17 homologous polypeptides and therapeutic uses thereof
INVENTOR(S): Chen, Jian, Princeton, NJ, UNITED STATES
Filvaroff, Ellen, San Francisco, CA, UNITED STATES
Fong, Sherman, Alameda, CA, UNITED STATES
Goddard, Audrey, San Francisco, CA, UNITED STATES
Godowski, Paul, Hillsborough, CA, UNITED STATES
Grimaldi, Christopher, San Francisco, CA, UNITED STATES
Gurney, Austin, Belmont, CA, UNITED STATES
Li, Hanzhong, San Mateo, CA, UNITED STATES
Hillan, Kenneth, San Francisco, CA, UNITED STATES
Tumas, Daniel, Orinda, CA, UNITED STATES

VanLookeren, Menno, San Francisco, CA, UNITED STATES
Vandlen, Richard, Hillsborough, CA, UNITED STATES
Watanabe, Colin, Moraga, CA, UNITED STATES
Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES
Wood, William I., Hillsborough, CA, UNITED STATES
Yansura, Daniel, Pacifica, CA, UNITED STATES
PATENT ASSIGNEE(S): Genentech, Inc., South San Francisco, CA, UNITED
STATES, 94080 (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003180255	A1	20030925
APPLICATION INFO.:	US 2003-410927	A1	20030409 (10)

RELATED APPLN. INFO.: Division of Ser. No. US 2001-816744, filed on 22 Mar
2001, GRANTED, Pat. No. US 6579520 Continuation-in-part
of Ser. No. WO 2001-US6520, filed on 28 Feb 2001,
PENDING Continuation-in-part of Ser. No. US
2000-747259, filed on 20 Dec 2000, GRANTED, Pat. No. US
6569645 Continuation-in-part of Ser. No. WO
2000-US23328, filed on 24 Aug 2000, PENDING
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: GENENTECH, INC., 1 DNA WAY, SOUTH SAN FRANCISCO, CA,
94080
NUMBER OF CLAIMS: 60
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 48 Drawing Page(s)
LINE COUNT: 8657
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention is directed to novel polypeptides having sequence
identity with IL-17, IL-17 receptors and to nucleic acid molecules
encoding those polypeptides. Also provided herein are vectors and host
cells comprising those nucleic acid sequences, chimeric polypeptide
molecules comprising the polypeptides of the present invention fused to
heterologous polypeptide sequences, antibodies which bind to the
polypeptides of the present invention and to methods for producing the
polypeptides of the present invention. Further provided herein are
methods for treating degenerative cartilaginous disorders and other
inflammatory diseases.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 18 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:237907 USPATFULL
TITLE: Compositions and methods for the therapy and diagnosis
of colon cancer
INVENTOR(S): King, Gordon E., Shoreline, WA, UNITED STATES
Meagher, Madeleine Joy, Seattle, WA, UNITED STATES
Xu, Jiangchun, Bellevue, WA, UNITED STATES
Secrist, Heather, Seattle, WA, UNITED STATES
Jiang, Yuqiu, Kent, WA, UNITED STATES
PATENT ASSIGNEE(S): Corixa Corporation, Seattle, WA, UNITED STATES, 98104
(U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003166064	A1	20030904
APPLICATION INFO.:	US 2002-99926	A1	20020314 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-33528, filed
on 26 Dec 2001, PENDING Continuation-in-part of Ser.
No. US 2001-920300, filed on 31 Jul 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-302051P	20010629 (60)
	US 2001-279763P	20010328 (60)
	US 2000-223283P	20000803 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH
AVE, SUITE 6300, SEATTLE, WA, 98104-7092
NUMBER OF CLAIMS: 17
EXEMPLARY CLAIM: 1
LINE COUNT: 8531
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Compositions and methods for the therapy and diagnosis of cancer,

particularly colon cancer, are disclosed. Illustrative compositions
comprise one or more colon tumor polypeptides, immunogenic portions
thereof, polynucleotides that encode such polypeptides, antigen
presenting cell that expresses such polypeptides, and T cells that are
specific for cells expressing such polypeptides. The disclosed
compositions are useful, for example, in the diagnosis, prevention
and/or treatment of diseases, particularly colon cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 19 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:201388
USPATFULL
TITLE: Combined methods for tumor vasculature coagulation and
treatment
INVENTOR(S): Thorpe, Philip E., Dallas, TX, UNITED STATES
King, Steven W., Rancho Santa Margarita, CA, UNITED
STATES
Gottstein, Claudia, Dallas, TX, UNITED STATES
PATENT ASSIGNEE(S): Board of Regents, The University of Texas System and
Peregrine Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003139374	A1	20030724
APPLICATION INFO.:	US 2002-259236	A1	20020927 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-325532P	20010927 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Shelley P.M. Fussey, Williams, Morgan & Amerson, P.C.,
Suite 250, 7676 Hillmont, Houston, TX, 77040
NUMBER OF CLAIMS: 43
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 10003
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are various defined combinations of agents for use in improved
anti-vascular therapies and coagulative tumor treatment. Particularly
provided are combined treatment methods, and associated compositions,
pharmaceuticals, medicaments, kits and uses, which together function
surprisingly effectively in the treatment of vascularized tumors. The
invention preferably involves a component or treatment step that
enhances the effectiveness of therapy using targeted or non-targeted
coagulants to cause tumor vasculature thrombosis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 20 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:187407
USPATFULL
TITLE: Combined methods for tumor vasculature coaguligand
treatment
INVENTOR(S): Thorpe, Philip E., Dallas, TX, UNITED STATES
King, Steven W., Rancho Santa Margarita, CA, UNITED
STATES
Gottstein, Claudia, Dallas, TX, UNITED STATES
PATENT ASSIGNEE(S): Board of Regents, The University of Texas System and
Peregrine Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003129193	A1	20030710
APPLICATION INFO.:	US 2002-259227	A1	20020927 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-325532P	20010927 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Shelley P.M. Fussey, Williams, Morgan & Amerson, P.C.,
Suite 250, 7676 Hillmont, Houston, TX, 77040
NUMBER OF CLAIMS: 45
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 10012
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are various defined combinations of agents for use in improved
anti-vascular therapies and coagulative tumor treatment. Particularly

provided are combined treatment methods, and associated compositions,
pharmaceuticals, medicaments, kits and uses, which together function
surprisingly effectively in the treatment of vascularized tumors. The
invention preferably involves a component or treatment step that
enhances the effectiveness of therapy using targeted or non-targeted
coagulants to cause tumor vasculature thrombosis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 21 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:180305
USPATFULL
TITLE: Combined compositions for tumor vasculature coaguligand
treatment
INVENTOR(S): Thorpe, Philip E., Dallas, TX, UNITED STATES
King, Steven W., Rancho Santa Margarita, CA, UNITED
STATES
Gottstein, Claudia, Dallas, TX, UNITED STATES
PATENT ASSIGNEE(S): Board of Regents, The University of Texas System (U.S.
corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003124132	A1	20030703
APPLICATION INFO.:	US 2002-259223	A1	20020927 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-325532P	20010927 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Shelley P.M. Fussey, Ph.D., WILLIAMS, MORGAN & AMERSON,
P.C., Suite 1100, 10333 Richmond Avenue, Houston, TX,
77042
NUMBER OF CLAIMS: 45
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 10025
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are various defined combinations of agents for use in improved
anti-vascular therapies and coagulative tumor treatment. Particularly
provided are combined treatment methods, and associated compositions,
pharmaceuticals, medicaments, kits and uses, which together function
surprisingly effectively in the treatment of vascularized tumors. The
invention preferably involves a component or treatment step that
enhances the effectiveness of therapy using targeted or non-targeted
coagulants to cause tumor vasculature thrombosis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 22 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:165436
USPATFULL
TITLE: Cocoa extract compounds and methods for making and
using the same
INVENTOR(S): Romanczyk,, Leo J., JR., Hackettstown, NJ, UNITED
STATES
Schmitz, Harold H., Branchburg, NJ, UNITED STATES
PATENT ASSIGNEE(S): MARS Incorporated (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003113290	A1	20030619
APPLICATION INFO.:	US 2002-127817	A1	20020422 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-776649, filed on 5 Feb
2001, PENDING Continuation of Ser. No. US 1997-831245,
filed on 2 Apr 1997, GRANTED, Pat. No. US 6297273
Continuation-in-part of Ser. No. US 1996-631661, filed
on 2 Apr 1996, ABANDONED Continuation of Ser. No. US
2000-717893, filed on 21 Nov 2000, PENDING Continuation
of Ser. No. US 1997-831245, filed on 2 Apr 1997,
GRANTED, Pat. No. US 6297273 Continuation-in-part of
Ser. No. US 1996-631661, filed on 2 Apr 1996, ABANDONED
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: CLIFFORD CHANCE US LLP, 200 PARK AVENUE, NEW YORK, NY,
10166
NUMBER OF CLAIMS: 208
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 258 Drawing Page(s)
LINE COUNT: 6136

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Disclosed and claimed are cocoa extracts, compounds, combinations thereof and compositions containing the same, such as polyphenols or procyanidins, methods for preparing such extracts, compounds and compositions, as well as uses for them, especially a polymeric compound of the formula A.sub.n, wherein A is a monomer of the formula:

##STR1##

wherein n is an integer from 2 to 18, such that there is at least one terminal monomeric unit A, and one or a plurality of additional monomeric units;

R is 3-(.alpha.)--OH, 3-(.beta.)--OH, 3-(.alpha.)--O-sugar, or 3-(.beta.)--O-sugar;

bonding between adjacent monomers takes place at positions 4, 6 or 8;

a bond of an additional monomeric unit in position 4 has alpha or beta stereochemistry;

X, Y and Z are selected from the group consisting of monomeric unit A, hydrogen, and a sugar, with the provisos that as to the at least one terminal monomeric unit, bonding of the additional monomeric unit thereto (the bonding of the additional monomeric unit adjacent to the terminal monomeric unit) is at position 4 and optionally Y=Z=hydrogen;

the sugar is optionally substituted with a phenolic moiety, at any position on the sugar, for instance via an ester bond, and

pharmaceutically acceptable salts or derivatives thereof (including oxidation products).

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 23 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:106233 USPATFULL
TITLE: Compositions and methods for the therapy and diagnosis of pancreatic cancer
INVENTOR(S): Benson, Darin R., Seattle, WA, UNITED STATES
Kalos, Michael D., Seattle, WA, UNITED STATES
Lodes, Michael J., Seattle, WA, UNITED STATES
Persing, David H., Redmond, WA, UNITED STATES
Hepler, William T., Seattle, WA, UNITED STATES
Jiang, Yuqiu, Kent, WA, UNITED STATES
PATENT ASSIGNEE(S): Corixa Corporation, Seattle, WA, UNITED STATES, 98104 (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003073144	A1	20030417
APPLICATION INFO.:	US 2002-60036	A1	20020130 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-333626P	20011127 (60)
	US 2001-305484P	20010712 (60)
	US 2001-265305P	20010130 (60)
	US 2001-267568P	20010209 (60)
	US 2001-313999P	20010820 (60)
	US 2001-291631P	20010516 (60)
	US 2001-287112P	20010428 (60)
	US 2001-278651P	20010321 (60)
	US 2001-265682P	20010131 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH AVE, SUITE 6300, SEATTLE, WA, 98104-7092
NUMBER OF CLAIMS: 17
EXEMPLARY CLAIM: 1
LINE COUNT: 14253
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compositions and methods for the therapy and diagnosis of cancer, particularly pancreatic cancer, are disclosed. Illustrative compositions comprise one or more pancreatic tumor polypeptides, immunogenic portions thereof, polynucleotides that encode such polypeptides, antigen presenting cell that expresses such polypeptides, and T cells that are specific for cells expressing such polypeptides. The disclosed compositions are useful, for example, in the diagnosis, prevention and/or treatment of diseases, particularly pancreatic cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 24 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:78522 USPATFULL
TITLE: IL-17 homologous polypeptides and therapeutic uses
thereof
INVENTOR(S): Chen, Jian, Princeton, NJ, UNITED STATES
Filvaroff, Ellen, San Francisco, CA, UNITED STATES
Fong, Sherman, Alameda, CA, UNITED STATES
Goddard, Audrey, San Francisco, CA, UNITED STATES
Godowski, Paul J., HIllsborough, CA, UNITED STATES
Grimaldi, J. Christopher, San Francisco, CA, UNITED
STATES
Gurney, Austin, Belmont, CA, UNITED STATES
Li, Hanzhong, San Mateo, CA, UNITED STATES
Hillan, Kenneth, San Francisco, CA, UNITED STATES
Tumas, Daniel, Orinda, CA, UNITED STATES
VanLookeren, Menno, San Francisco, CA, UNITED STATES
Vandlen, Richard, Hillsborough, CA, UNITED STATES
Watanabe, Colin K., Moraga, CA, UNITED STATES
Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES
Wood, William I., Hillsborough, CA, UNITED STATES
Yansura, Daniel, Pacifica, CA, UNITED STATES
PATENT ASSIGNEE(S): GENENTECH, INC. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003054442	A1	20030320
APPLICATION INFO.:	US 2001-908827	A1	20010718 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1999-311832, filed on 14
May 1999, PENDING Continuation of Ser. No. US
1999-380138, filed on 25 Aug 1999, PENDING Continuation
of Ser. No. US 1999-380142, filed on 25 Aug 1999,
ABANDONED Continuation of Ser. No. US 2000-644848,
filed on 22 Aug 2000, PENDING Continuation of Ser. No.
US 2000-747259, filed on 20 Dec 2000, PENDING
Continuation of Ser. No. US 2001-816744, filed on 22
Mar 2001, PENDING Continuation of Ser. No. US
2001-854208, filed on 10 May 2001, PENDING Continuation
of Ser. No. US 2001-854280, filed on 10 May 2001,
PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	WO 1999-US5028	19990308
	WO 1999-US10733	19990514
	WO 1999-US31274	19991230
	WO 2000-US4341	20000218
	WO 2000-US5601	20000301
	WO 2000-US5841	20000302
	WO 2000-US7532	20000321
	WO 2000-US15264	20000602
	WO 2000-US23328	20000824
	WO 2000-US30873	20001110
	WO 2000-US32678	20001201
	WO 2000-US34956	20001220
	WO 2001-US6520	20010228
	US 1998-85579P	19980515 (60)
	US 1998-113621P	19981223 (60)
	US 1999-130232P	19990421 (60)
	US 1999-131022P	19990426 (60)
	US 1999-134287P	19990514 (60)
	US 1999-138387P	19990609 (60)
	US 1999-172096P	19991223 (60)
	US 2000-175481P	20000111 (60)
	US 2000-191007P	20000321 (60)
	US 2000-213807P	20000622 (60)
	US 2000-242837P	20001024 (60)
	US 2000-244072P	20001026 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: GENENTECH, INC., 1 DNA WAY, SOUTH SAN FRANCISCO, CA, 94080
NUMBER OF CLAIMS: 60
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 47 Drawing Page(s)
LINE COUNT: 8091
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention is directed to novel polypeptides and to nucleic
acid molecules encoding those polypeptides. Also provided herein are
vectors and host cells comprising those nucleic acid sequences, chimeric
polypeptide molecules comprising the polypeptides of the present

invention fused to heterologous polypeptide sequences, antibodies which
bind to the polypeptides of the present invention and to methods for
producing the polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 25 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:11111 USPATFULL
TITLE: IL-17 homologous polypeptides and therapeutic uses thereof
INVENTOR(S): Chen, Jian, Princeton, NJ, UNITED STATES
Filvaroff, Ellen, San Francisco, CA, UNITED STATES
Fong, Sherman, Alameda, CA, UNITED STATES
Goddard, Audrey, San Francisco, CA, UNITED STATES
Godowski, Paul J., Burlingame, CA, UNITED STATES
Grimaldi, Christopher, San Francisco, CA, UNITED STATES
Gurney, Austin L., Belmont, CA, UNITED STATES
Li, Hanzhong, San Mateo, CA, UNITED STATES
Hillan, Kenneth, San Francisco, CA, UNITED STATES
Tumas, Daniel, Orinda, CA, UNITED STATES
VanLookeren, Menno, San Francisco, CA, UNITED STATES
Vandlen, Richard, Hillsborough, CA, UNITED STATES
Watanabe, Colin, Moraga, CA, UNITED STATES
Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES
Wood, William I., Hillsborough, CA, UNITED STATES
Yansura, Daniel G., Pacifica, CA, UNITED STATES
PATENT ASSIGNEE(S): GENENTECH, INC. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003008815	A1	20030109
	US 6569645	B2	20030527
APPLICATION INFO.:	US 2000-747259	A1	20001220 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1999-311832, filed on 14 May 1999, PENDING
Continuation-in-part of Ser. No. US 2000-644848, filed on 22 Aug 2000, PENDING
Continuation-in-part of Ser. No. WO 2000-US4341, filed on 18 Feb 2000, UNKNOWN
Continuation-in-part of Ser. No. WO 2000-US23328, filed on 24 Aug 2000, UNKNOWN
Continuation-in-part of Ser. No. WO 2000-US32678, filed on 1 Dec 2000, UNKNOWN
Continuation-in-part of Ser. No. WO 1999-US31274, filed on 30 Dec 1999, UNKNOWN
Continuation-in-part of Ser. No. WO 2000-US7532, filed on 21 Mar 2000, UNKNOWN
Continuation-in-part of Ser. No. WO 2000-US5841, filed on 2 Mar 2000, UNKNOWN
Continuation-in-part of Ser. No. WO 2000-US15264, filed on 2 Jun 2000, UNKNOWN
Continuation-in-part of Ser. No. WO 2000-US30873, filed on 10 Nov 2000, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-253646P	20001128 (60)
	US 1999-172096P	19991223 (60)
	US 2000-175481P	20000111 (60)
	US 2000-191007P	20000321 (60)
	US 2000-213087P	20000620 (60)
	US 2000-242837P	20001024 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: GENENTECH, INC., 1 DNA WAY, SOUTH SAN FRANCISCO, CA, 94080
NUMBER OF CLAIMS: 60
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 47 Drawing Page(s)
LINE COUNT: 8685
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention is directed to novel polypeptides and to nucleic
acid molecules encoding those polypeptides. Also provided herein are
vectors and host cells comprising those nucleic acid sequences, chimeric
polypeptide molecules comprising the polypeptides of the present
invention fused to heterologous polypeptide sequences, antibodies which
bind to the polypeptides of the present invention and to methods for
producing the polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 26 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:3511 USPATFULL
TITLE: IL-17 homologous polypeptides and therapeutic uses
thereof
INVENTOR(S): Chen, Jian, Princeton, NJ, UNITED STATES
Filvaroff, Ellen, San Francisco, CA, UNITED STATES
Fong, Sherman, Alameda, CA, UNITED STATES
Goddard, Audrey, San Francisco, CA, UNITED STATES
Godowski, Paul, Burlingame, CA, UNITED STATES
Grimaldi, Christopher, San Francisco, CA, UNITED STATES
Gurney, Austin, Belmont, CA, UNITED STATES
Li, Hanzhong, San Mateo, CA, UNITED STATES
Hillan, Kenneth, San Francisco, CA, UNITED STATES
Tumas, Daniel, Orinda, CA, UNITED STATES
VanLookeren, Menno, San Francisco, CA, UNITED STATES
Vandlen, Richard, Hillsborough, CA, UNITED STATES
Watanabe, Colin, Moraga, CA, UNITED STATES
Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES
Wood, William I., Hillsborough, CA, UNITED STATES
Yansura, Daniel, Pacifica, CA, UNITED STATES
PATENT ASSIGNEE(S): GENENTECH, INC. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003003546	A1	20030102
	US 6579520	B2	20030617
APPLICATION INFO.:	US 2001-816744	A1	20010322 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1999-311832, filed
on 14 May 1999, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	WO 2001-US6520	20010228
	WO 2000-US34956	20001220
	WO 2000-US32678	20001201
	WO 2000-US30873	20001110
	WO 2000-US23328	20000824
	WO 2000-US15264	20000602
	WO 2000-US7532	20000321
	WO 2000-US5841	20000302
	WO 2000-US5601	20000301
	WO 2000-US4341	20000218
	WO 1999-US31274	19991230
	WO 1999-US10733	19990514
	WO 1999-US5028	19990308
	US 2000-253646P	20001128 (60)
	US 2000-244072P	20001026 (60)
	US 2000-242837P	20001024 (60)
	US 2000-213807P	20000622 (60)
	US 2000-191007P	20000321 (60)
	US 2000-175481P	20000111 (60)
	US 1999-172096P	19991223 (60)
	US 1999-138387P	19990609 (60)
	US 1999-134287P	19990514 (60)
	US 1999-131022P	19990426 (60)
	US 1999-130232P	19990421 (60)
	US 1998-113621P	19981223 (60)
	US 1998-85579P	19980515 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: GENENTECH, INC., 1 DNA WAY, SOUTH SAN FRANCISCO, CA, 94080
NUMBER OF CLAIMS: 60
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 48 Drawing Page(s)
LINE COUNT: 7774
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention is directed to novel polypeptides having sequence identity with IL-17, IL-17 receptors and to nucleic acid molecules encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide molecules comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention. Further provided herein are methods for treating degenerative cartilaginous disorders and other inflammatory diseases.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 27 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:337302 USPATFULL
TITLE: Cocoa extract compounds and methods for making and
using the same

INVENTOR(S): Romanczyk, Jr., Leo J., Hackettstown, NJ, United States
Hammerstone, Jr., John F., Nazareth, PA, United States
Buck, Margaret M., Morristown, NJ, United States
Post, Laurie S., Great Meadows, NJ, United States
Cipolla, Giovanni G., Alpha, NJ, United States
McClelland, Craig A., East Stroudsburg, PA, United States
Mundt, Jeff A., Hackettstown, NJ, United States
Schmitz, Harold H., Branchburg, NJ, United States
PATENT ASSIGNEE(S): Mars Incorporated, McLean, VA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6670390	B1	20031230
APPLICATION INFO.:	US 2000-717893		20001121 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1997-831245, filed on 2 Apr 1997, now patented, Pat. No. US 6297273
Continuation-in-part of Ser. No. US 1996-631661, filed on 2 Apr 1996, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Solola, T. A.
LEGAL REPRESENTATIVE: Kelley, Margaret B., Clifford Chance Rogers & Wells
NUMBER OF CLAIMS: 26
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 248 Drawing Figure(s); 232 Drawing Page(s)
LINE COUNT: 4609
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed and claimed are cocoa extracts, compounds, combinations
thereof and compositions containing the same, such as polyphenols or
procyanidins, methods for preparing such extracts, compounds and
compositions, as well as uses for them, especially a polymeric compound
of the formula A.sub.n, wherein A is a monomer of the formula: ##STR1##

wherein

n is an integer from 2 to 18, such that there is at least one terminal
monomeric unit A, and one or a plurality of additional monomeric units;

R is 3-(.alpha.)-OH, 3-(.beta.)-OH, 3-(.alpha.)-O-sugar, or
3-(.beta.)-O-sugar.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 28 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2003:26240 USPATFULL
TITLE: Methods of treating nitric oxide and cytokine mediated disorders
INVENTOR(S): Singh, Inderjit, Mount Pleasant, SC, United States
PATENT ASSIGNEE(S): Medical University of South Carolina, Charleston, SC, United States (U.S. corporation)
MUSC Foundation for Research Development, Charleston, SC, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6511800	B1	20030128
APPLICATION INFO.:	US 2000-579791		20000525 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. WO 1998-US25360, filed on 25 Nov 1998

	NUMBER	DATE
PRIORITY INFORMATION:	US 1997-66839P	19971125 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Gitomer, Ralph
LEGAL REPRESENTATIVE: Fulbright & Jaworski LLP
NUMBER OF CLAIMS: 50
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 16 Drawing Figure(s); 11 Drawing Page(s)
LINE COUNT: 7562
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The current invention discloses novel methods for the inhibition of
inducible nitric oxide synthesis (iNOS) and the production of NO.
Methods of inhibiting the induction of proinflammatory cytokines are
also described. Methods of treating various disease states, such as
X-linked adrenoleukodystrophy, multiple sclerosis, Alzheimer's and

septic shock using inhibitors of iNOS and cytokine induction are disclosed. The inhibitors include the exemplary compounds lovastatin, a sodium salt of phenylacetic acid (NaPA), FPT inhibitor II, N-acetyl cysteine (NAC), and cAMP. Methods of treating a nitric oxide or cytokine mediated disorder in a cell comprising administering a biologically effective amount of at least one induction suppressor of an inducible nitric oxide synthase or a cytokine is also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 29 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 2
ACCESSION NUMBER: 2003:908561 CAPLUS
DOCUMENT NUMBER: 140:192445
TITLE: Ifosfamide impairs the allostimulatory capacity of human dendritic cells by intracellular glutathione depletion
AUTHOR(S): Kuppner, Maria C.; Scharner, Anabel; Milani, Valeria; von Hesler, Christoph; Tschoep, Katharina E.; Heinz, Oksana; Issels, Rolf D.
CORPORATE SOURCE: Klinikum Grosshadem, Medical Clinic III, Ludwig-Maximillians-University, Munich, Germany
SOURCE: Blood (2003), 102(10), 3668-3674
CODEN: BLOOAW; ISSN: 0006-4971
PUBLISHER: American Society of Hematology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Ifosfamide, a clin. potent chemotherapeutic agent, causes the depletion of intracellular glutathione (GSH) levels in various cell types. GSH is the major intracellular reductant against oxidative stress. 4-Hydroxyifosfamide (4-OH-IF), the activated form of ifosfamide, depletes GSH levels in T cells and natural killer (NK) cells; this is accompanied by a decrease in T-cell and NK-cell function. Here we demonstrate for the first time that human monocyte-derived dendritic cells (DCs) express higher constitutive levels of GSH and are less sensitive to 4-OH-IF-induced GSH depletion than T cells and NK cells. Treatment of DCs with 4-OH-IF significantly reduced their ability to stimulate allogeneic T-cell proliferation and interferon-.gamma. (IFN-.gamma.) prodn. Ifosfamide also decreased DC interleukin-12p70 (IL-12p70) prodn. after stimulation with lipopolysaccharide (LPS) and IFN-.gamma.. The decrease in allostimulatory capacity and in IFN-.gamma. and ***IL*** - ***12*** prodn. correlated with a decrease in intracellular GSH in the DCs. The responses could be restored by reconstituting DC GSH levels with glutathione monoethyl ester (GSH-OEt). 4-OH-IF had ***no*** ***inhibitory*** effect on the ability of DCs to present exogenously added tyrosinase peptide to tyrosinase-specific cytotoxic T lymphocytes (CTLs). These studies suggest that in cancer patients treated with ifosfamide, protection strategies based on glutathione reconstitution may enhance DC function.
REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 30 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 3
ACCESSION NUMBER: 2003:970254 CAPLUS
DOCUMENT NUMBER: 140:123097
TITLE: Unique regulation profile of prostaglandin E1 on adhesion molecule expression and cytokine production in human peripheral blood mononuclear cells
AUTHOR(S): Takahashi, Hideo Kohka; Iwagaki, Hiromi; Tamura, Ryuji; Xue, Dong; Sano, Masahiro; Mori, Shuji; Yoshino, Tadashi; Tanaka, Noriaki; Nishibori, Masahiro
CORPORATE SOURCE: Department of Pharmacology, Okayama University Graduate School of Medicine and Dentistry, Okayama, Japan
SOURCE: Journal of Pharmacology and Experimental Therapeutics (2003), 307(3), 1188-1195
CODEN: JPETAB; ISSN: 0022-3565
PUBLISHER: American Society for Pharmacology and Experimental Therapeutics
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In the present study, the authors examd. the effects of prostaglandin E1

(PGE1) on the expression of intercellular adhesion mol. (ICAM)-1, B7.1,
B7.2, CD40, and CD40 ligand (CD40L) on peripheral blood mononuclear cells
(PBMC) using fluorescence-activated cell sorting anal. as well as its
effects on cytokine prodn. using ELISA. Whereas ***no***
inhibitor of spontaneous expression of adhesion mols. was
reported, the authors found that PGE1 inhibited spontaneous ICAM-1, B7.2,
and CD40 expression on monocytes in a concn.-dependent manner but had no
effect on the expression of B7.1 and CD40L. Although interleukin (IL)-18
induced the expression of ICAM-1, B7.2, CD40, and CD40L, PGE1 prevented
IL-18-induced expression of ICAM-1, B7.2, and CD40. The authors examd.
the involvement of five subtypes of PGE1 receptors (IP, EP1, EP2, EP3, and
EP4) in the effect of PGE1 on the expression of these adhesion mols. using
subtype-specific agonists. Among EP receptor agonists, EP2 and EP4
receptor agonists inhibited IL-18-elicited ICAM-1, B7.2, and CD40
expression. ONO-1301 (IP receptor agonist) prevented the expression of
ICAM-1, B7.2, and CD40 regardless of the presence of IL-18 with the same
potency as PGE1. The effect of a combination of ONO-1301 and 11-deoxy
(D)-PGE1 (EP2/EP4 receptor agonist) on ICAM-1, B7.2, and CD40 expression
mimicked that of PGE1. Moreover, PGE1 inhibited the prodn. of ***IL***
- ***12*** and interferon-.gamma. in PBMC in the presence and absence
of IL-18, whereas PGE1 induced IL-10 prodn. In conclusion, IP receptor
and EP2/EP4 receptor play an important role in the action of PGE1 on the
expression of adhesion mols. on monocytes and cytokine prodn.
REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 31 OF 74 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
ACCESSION NUMBER: 2003056465 EMBASE
TITLE: Cytotoxicity in glioma cells due to ***interleukin*** - ***12*** and interleukin-18-stimulated macrophages mediated by interferon-.gamma.-regulated nitric oxide.
AUTHOR: Kito T.; Kuroda E.; Yokota A.; Yamashita U.
CORPORATE SOURCE: Dr. U. Yamashita, Department of Immunology, Univ. of Occup./Environmental Health, School of Medicine, 1-1 Iseigaoka, Yahatanishi-ku, Kitakyusyu 807-8555, Japan.
yama-uki@med.uoeh-u.ac.jp
SOURCE: Journal of Neurosurgery, (1 Feb 2003) 98/2 (385-392).
Refs: 45
ISSN: 0022-3085 CODEN: JONSAC
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 008 Neurology and Neurosurgery
026 Immunology, Serology and Transplantation
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Object. Interleukin (***IL***)- ***12*** and IL-18 synergistically
mediate antitumor responses through the production of interferon-.gamma.
(IFN.gamma.) by T and natural killer (NK) cells. Recently, it has been
reported that macrophages stimulated with these cytokines also produce
IFN.gamma., which led the authors to investigate the antiglioma activity
of macrophages stimulated by the combination of these cytokines in vitro.
Methods. Dish-adherent peritoneal exudate cells, which had been elicited
in thioglycollate broth as a source of macrophages, were used in the
experiment. The murine glioma cell lines VM-glioma and 203G were labeled
with [(3)H]thymidine for a cytotoxicity assay of macrophages. In response
to the combined stimulation by ***IL*** - ***12*** and IL-18,
macrophages expressed potent cytotoxic activity against glioma cells in
association with increasing production of IFN.gamma. and nitric oxide (***NO***). ***Inhibitors*** of NO abrogated the cytotoxic activity
of the macrophages, which had been induced by IL-12 and IL-18, despite the
increase in IFN.gamma. production. Neutralization of IFN.gamma. or use of
macrophages obtained from IFN.gamma. gene-knockout mice markedly reduced
not only cytotoxic activity, but also NO production. Depletion of T and NK
cells from the macrophage population, which was achieved using antibody
plus complement treatment, slightly reduced macrophage activities,

suggesting that these are the main effector cells, although T and NK cells
may partially participate in this cytotoxicity. Conclusions. Macrophages
stimulated with ***IL*** - ***12*** and IL-18 produced IFN.gamma.
and NO, which in turn mediated the antiglioma response. Therefore,
macrophages as well as T and NK cells play an important role in antitumor
responses stimulated by ***IL*** - ***12*** and IL-18.

L5 ANSWER 32 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 4
ACCESSION NUMBER: 2003:514704 CAPLUS
DOCUMENT NUMBER: 139:270480
TITLE: Antitumor effects of yeast (NBG, Immutol) and their
clinical significance
AUTHOR(S): Yagita, Akikuni; Maruyama, Shoji; Sukegawa, Yasushi;
Takenoshita, Seiichi; Kanazawa, Masashi; Katoh, Ryoji;
Hagi, Hiroo; Fujimoto, Shigeyoshi
CORPORATE SOURCE: Institute of Immunotherapy for Cancer, Kinki
University, Japan
SOURCE: Biotherapy (Tokyo, Japan) (2003), 17(3), 257-266
CODEN: BITPE9; ISSN: 0914-2223
PUBLISHER: Gan to Kagaku Ryohosha
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB The antineoplastic effect of .beta.-1,3/1,6 glucan (basidiomycete prepn.)
is well known. However, the antineoplastic effect of yeast-derived
.beta.-1,3/1,6 glucan, such as NBG (Immutol), has been little
investigated. In the present study, we used models of 3LL s.c. tumor
grafts in Th1 strain B10 mice (high tumor resistance) and colon 26 s.c.
tumor grafts in Th2 strain BALB/c mice (low tumor resistance) to
investigate the endogenous ***interleukin*** - ***12*** (***IL***
- ***12***) induction ability and antineoplastic effect of NBG. The
results showed antitumor activity in the NBG-administered groups of both
B10 and BALB/c mice, but in terms of endogenous ***IL*** - ***12***
a significantly high productivity was seen in the highly tumor resistant
B10 mice only. No significant difference was seen in the BALB/c mice,
which have a low tumor resistance. NBG (Immutol) was administered for 3
mo or longer to more than 260 patients with advanced or terminal stage
cancer, in whom effects from immunotherapy are not conventionally seen.
The immunol. changes and clin. efficacy were investigated. With addnl.
administration of NBG (Immutol, 6T or more/day), there was a marked
increase in the no. of NK cells, followed by an increase in NKT cells.
While significant differences in ***IL*** - ***12*** productivity
were not seen in all patients, a significant difference was seen when a
comparison was made in effective cases (96 cases) only. The productivity
of interleukin-10 (IL-10), one of the major immunosuppressant cytokines,
was significantly inhibited after the administration of NBG. However,
no ***inhibitory*** action on the angiogenesis promotion
factor VEGF was obsd.

L5 ANSWER 33 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 5
ACCESSION NUMBER: 2002:309776 CAPLUS
DOCUMENT NUMBER: 136:319388
TITLE: Methods and compositions for enhancing the
immunostimulatory effect of ***interleukin*** -
12
INVENTOR(S): Trinchieri, Giorgio; Lee, William M. F.; Koblish,
Holly
PATENT ASSIGNEE(S): The Wistar Institute of Anatomy and Biology, USA; The
Trustees of the University of Pennsylvania
SOURCE: U.S., 19 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6375944	B1	20020423	US 1999-395038	19990913
US 2002081277	A1	20020627	US 2002-79068	20020220

PRIORITY APPLN. INFO.: US 1998-101698P P 19980925
US 1999-395038 A3 19990913
AB The invention discloses a method for enhancing the therapeutic and
adjuvant use of ***IL*** - ***12*** by reducing unwanted transient

immunosuppression caused by ***IL*** - ***12*** or by high doses
thereof by co-administering ***IL*** - ***12*** with an effective
amt. of an agent that inhibits or neutralizes nitric oxide (NO) in vivo.
This enhanced vaccine therapy involves co-administering the ***IL*** -
12 adjuvant, a selected vaccine antigen and the ***NO***
inhibiting /neutralizing agent. Addnl., the toxicity of ***IL***
- ***12*** treatment may be reduced by co-administering ***IL*** -
12 with an effective amt. of the ***NO*** ***inhibiting***
or neutralizing agent. A therapeutic compn. characterized by reduced
toxicity in mammals contains ***IL*** - ***12*** , preferably a low
dose thereof, and an ***NO*** ***inhibiting*** or neutralizing
agent in a pharmaceutically acceptable carrier. A vaccine compn. contains
an effective adjuvant amt. of ***IL*** - ***12*** , an effective amt.
of an ***NO*** ***inhibiting*** or neutralizing agent, and an
effective protective amt. of a vaccine antigen in a pharmaceutically
acceptable carrier.
REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 34 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2002:322509 USPATFULL
TITLE: IL-17 homologous polypedies and therapeutic uses
thereof
INVENTOR(S): Chen, Jian, Princeton, NJ, UNITED STATES
Filvaroff, Ellen, San Francisco, CA, UNITED STATES
Fong, Sherman, Alameda, CA, UNITED STATES
French, Dorothy, Redwood City, CA, UNITED STATES
Goddard, Audrey, San Francisco, CA, UNITED STATES
Godowski, Paul J., Hillsborough, CA, UNITED STATES
Grimaldi, J. Christopher, San Francisco, CA, UNITED STATES
Gurney, Austin L., Belmont, CA, UNITED STATES
Hillan, Kenneth J., San Francisco, CA, UNITED STATES
Hymowitz, Sarah G., San Francisco, CA, UNITED STATES
Li, Hanzhong, San Mateo, CA, UNITED STATES
Pan, James, Zitobicoke, CANADA
Starovasnik, Melissa A., San Francisco, CA, UNITED STATES
Tumas, Daniel, Orinda, CA, UNITED STATES
Van Lookeren, Menno, San Francisco, CA, UNITED STATES
Vandlen, Richard, Hillsborough, CA, UNITED STATES
Watanabe, Colin K., Moraga, CA, UNITED STATES
Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES
Wood, William I., Hillsborough, CA, UNITED STATES
Yansura, Daniel G., Pacifica, CA, UNITED STATES
PATENT ASSIGNEE(S): GENENTECH, INC. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002182673	A1	20021205
APPLICATION INFO.:	US 2001-157	A1	20011030 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-931836, filed
on 16 Aug 2001, PENDING
Continuation-in-part of Ser.
No. US 2001-929404, filed on 13 Aug 2001, PENDING
Continuation-in-part of Ser. No. US 2001-918585, filed
on 30 Jul 2001, PENDING
Continuation-in-part of Ser.
No. US 2001-908827, filed on 18 Jul 2001, PENDING
Continuation-in-part of Ser. No. US 2001-874503, filed
on 5 Jun 2001, PENDING
Continuation-in-part of Ser. No.
US 2001-854280, filed on 10 May 2001, PENDING
Continuation-in-part of Ser. No. US 2001-854208, filed
on 10 May 2001, PENDING
Continuation-in-part of Ser.
No. US 2001-816744, filed on 22 Mar 2001, PENDING
Continuation-in-part of Ser. No. US 2000-747259, filed
on 20 Dec 2000, PENDING
Continuation-in-part of Ser.
No. US 2000-644848, filed on 22 Aug 2000, PENDING

Continuation-in-part of Ser. No. US 1999-380142, filed
on 25 Aug 1999, ABANDONED Continuation-in-part of Ser.
No. US 1999-380138, filed on 25 Aug 1999, PENDING
Continuation-in-part of Ser. No. US 1999-311832, filed
on 14 May 1999, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	WO 2001-US21735	20010709
	WO 2001-US21066	20010629
	WO 2001-US19692	20010620
	WO 2001-US17800	20010601
	WO 2001-US6520	20010228
	WO 2000-US34956	20001220
	WO 2000-US32678	20001201
	WO 2000-US30873	20001110
	WO 2000-US23328	20000824
	WO 2000-US15264	20000602
	WO 2000-US7532	20000321
	WO 2000-US5841	20000302
	WO 2000-US5601	20000301
	WO 2000-US4341	20000218
	WO 1999-US31274	19991230
	WO 1999-US10733	19990514
	WO 1999-US5028	19990308
	US 2000-253646P	20001128 (60)
	US 2000-244072P	20001026 (60)
	US 2000-242837P	20001024 (60)
	US 2000-213807P	20000622 (60)
	US 2000-191007P	20000321 (60)
	US 2000-175481P	20000111 (60)
	US 1999-172096P	19991223 (60)
	US 1999-138387P	19990609 (60)
	US 1999-134287P	19990514 (60)
	US 1999-131022P	19990426 (60)
	US 1999-130232P	19990421 (60)
	US 1998-113621P	19981223 (60)
	US 1998-85579P	19980515 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: GENENTECH, INC., 1 DNA WAY, SOUTH SAN FRANCISCO, CA, 94080
NUMBER OF CLAIMS: 60
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 70 Drawing Page(s)
LINE COUNT: 8889
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention is directed to novel polypeptides having sequence
identity with IL-17, IL-17 receptors and to nucleic acid molecules
encoding those polypeptides. Also provided herein are vectors and host
cells comprising those nucleic acid sequences, chimeric polypeptide
molecules comprising the polypeptides of the present invention fused to
heterologous polypeptide sequences, antibodies which bind to the
polypeptides of the present invention and to methods for producing the
polypeptides of the present invention. Further provided herein are
methods for treating degenerative cartilaginous disorders and other
inflammatory diseases.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 35 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2002:314711 USPATFULL
TITLE: IL-17 homologous polypeptides and therapeutic uses
thereof
INVENTOR(S): Chen, Jian, Princeton, NJ, UNITED STATES
Filvaroff, Ellen, San Francisco, CA, UNITED STATES
Fong, Sherman, Alameda, CA, UNITED STATES
Goddard, Audrey, San Francisco, CA, UNITED STATES
Godowski, Paul J., Burlingame, CA, UNITED STATES
Grimaldi, J. Christopher, San Francisco, CA, UNITED STATES
Gurney, Austin, Belmont, CA, UNITED STATES
Li, Hanzhong, San Mateo, CA, UNITED STATES
Hillan, Kenneth, San Francisco, CA, UNITED STATES
Hymowitz, Sarah G., San Francisco, CA, UNITED STATES
Tumas, Daniel, Orinda, CA, UNITED STATES
Starvovasnik, Melissa A., San Francisco, CA, UNITED STATES
Lookeren, Menno Van, San Francisco, CA, UNITED STATES
Vandlen, Richard, Hillsborough, CA, UNITED STATES
Watanabe, Colin, Moraga, CA, UNITED STATES
Williams, P. Mickey, Half Moon Bay, CA, UNITED STATES
Wood, William I., Hillsborough, CA, UNITED STATES
Yansura, Daniel G., Pacifica, CA, UNITED STATES
PATENT ASSIGNEE(S): GENENTECH, INC. (U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 2002177188 A1 20021128
APPLICATION INFO.: US 2001-874503 A1 20010605 (9)

NUMBER DATE

PRIORITY INFORMATION: WO 2001-US6520 20010228
WO 2000-US34956 20001220
WO 2000-US32678 20001201
WO 2000-US30873 20001110
WO 2000-US23328 20000824
WO 2000-US15264 20000602
WO 2000-US7532 20000321
WO 2000-US5841 20000302
WO 2000-US5601 20000301
WO 2000-US4341 20000218
WO 1999-US31274 19991230
WO 1999-US10733 19990514
WO 1999-US5028 19990308
US 2000-253646P 20001128 (60)
US 2000-244072P 20001026 (60)
US 2000-242837P 20001024 (60)
US 2000-213807P 20000622 (60)
US 2000-191007P 20000321 (60)
US 2000-175481P 20000111 (60)
US 1999-172096P 19991223 (60)
US 1999-138387P 19990609 (60)
US 1999-134287P 19990514 (60)
US 1999-131022P 19990426 (60)
US 1999-130232P 19990421 (60)
US 1998-113621P 19981223 (60)
US 1998-85579P 19980515 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: GENENTECH, INC., 1 DNA WAY, SOUTH SAN FRANCISCO, CA, 94080
NUMBER OF CLAIMS: 60
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 54 Drawing Page(s)
LINE COUNT: 8549
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention is directed to novel polypeptides having sequence identity with IL-17, IL-17 receptors and to nucleic acid molecules encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide molecules comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention. Further provided herein are methods for treating degenerative cartilaginous disorders and other inflammatory diseases.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 36 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2002:288103 USPATFULL
TITLE: Use of combretastatin A4 and its prodrugs as an immune enhancing therapy
INVENTOR(S): Pero, Ronald W., Sandgate, VT, UNITED STATES
Lee, Francis Y.F., Yardley, PA, UNITED STATES
Edvardsen, Klaus, Lund, SWEDEN
Sjogren, Hans Olov, Lund, SWEDEN

NUMBER KIND DATE

PATENT INFORMATION: US 2002160973 A1 20021031
US 6773702 B2 20040810
APPLICATION INFO.: US 2001-34746 A1 20011226 (10)

NUMBER DATE

PRIORITY INFORMATION: US 2000-258283P 20001226 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: COOPER & DUNHAM LLP, 1185 Ave. of the Americas, New York, NY, 10036
NUMBER OF CLAIMS: 14
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 9 Drawing Page(s)
LINE COUNT: 1160
AB A method of treating immune suppression in a warm-blooded animal bearing a tumor, by administering to the animal an amount of combretastatin A4 and/or a prodrug thereof effective to enhance immune responsiveness without causing vascular destruction. Immunotherapy treatment to inhibit or kill tumor cells includes administering to the animal an immune-response-stimulating agent such as a vaccine of tumor cells genetically modified to produce an immune-response-enhancing cytokine while counteracting tumor-induced immune suppression in the animal by administering combretastatin A4 and/or a prodrug thereof.

L5 ANSWER 37 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2002:272801
USPATFULL
TITLE: Compositions and methods for the therapy and diagnosis
of colon cancer
INVENTOR(S): Stolk, John A., Bothell, WA, UNITED STATES
Xu, Jiangchun, Bellevue, WA, UNITED STATES
Chenault, Ruth A., Seattle, WA, UNITED STATES
Meagher, Madeleine Joy, Seattle, WA, UNITED STATES
PATENT ASSIGNEE(S): Corixa Corporation, Seattle, WA, UNITED STATES, 98104
(U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002150922	A1	20021017
APPLICATION INFO.:	US 2001-998598	A1	20011116 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-304037P	20010710 (60)
	US 2001-279670P	20010328 (60)
	US 2001-267011P	20010206 (60)
	US 2000-252222P	20001120 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH
AVE, SUITE 6300, SEATTLE, WA, 98104-7092
NUMBER OF CLAIMS: 17
EXEMPLARY CLAIM: 1
LINE COUNT: 9233
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Compositions and methods for the therapy and diagnosis of cancer,
particularly colon cancer, are disclosed. Illustrative compositions
comprise one or more colon tumor polypeptides, immunogenic portions
thereof, polynucleotides that encode such polypeptides, antigen
presenting cell that expresses such polypeptides, and T cells that are
specific for cells expressing such polypeptides. The disclosed
compositions are useful, for example, in the diagnosis, prevention
and/or treatment of diseases, particularly colon cancer.

L5 ANSWER 38 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2002:243051
USPATFULL
TITLE: Compositions and methods for the therapy and diagnosis
of ovarian cancer
INVENTOR(S): Algate, Paul A., Issaquah, WA, UNITED STATES
Jones, Robert, Seattle, WA, UNITED STATES
Harlocker, Susan L., Seattle, WA, UNITED STATES
PATENT ASSIGNEE(S): Corixa Corporation, Seattle, WA, UNITED STATES, 98104
(U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002132237	A1	20020919
APPLICATION INFO.:	US 2001-867701	A1	20010529 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-207484P	20000526 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH
AVE, SUITE 6300, SEATTLE, WA, 98104-7092
NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 1
LINE COUNT: 25718
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Compositions and methods for the therapy and diagnosis of cancer,
particularly ovarian cancer, are disclosed. Illustrative compositions
comprise one or more ovarian tumor polypeptides, immunogenic portions
thereof, polynucleotides that encode such polypeptides, antigen
presenting cell that expresses such polypeptides, and T cells that are
specific for cells expressing such polypeptides. The disclosed
compositions are useful, for example, in the diagnosis, prevention
and/or treatment of diseases, particularly ovarian cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 39 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2002:242791 USPATFULL
TITLE: Compositions and methods for the therapy and diagnosis
of colon cancer
INVENTOR(S): King, Gordon E., Shoreline, WA, UNITED STATES
Meagher, Madeleine Joy, Seattle, WA, UNITED STATES
Xu, Jiangchun, Bellevue, WA, UNITED STATES
Secrist, Heather, Seattle, WA, UNITED STATES
PATENT ASSIGNEE(S): Corixa Corporation, Seattle, WA, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002131971	A1	20020919
APPLICATION INFO.:	US 2001-33528	A1	20011226 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-920300, filed on 31 Jul 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-302051P	20010629 (60)
	US 2001-279763P	20010328 (60)
	US 2000-223283P	20000803 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH AVE, SUITE 6300, SEATTLE, WA, 98104-7092
NUMBER OF CLAIMS: 17
EXEMPLARY CLAIM: 1
LINE COUNT: 8083
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Compositions and methods for the therapy and diagnosis of cancer, particularly colon cancer, are disclosed. Illustrative compositions comprise one or more colon tumor polypeptides, immunogenic portions thereof, polynucleotides that encode such polypeptides, antigen presenting cell that expresses such polypeptides, and T cells that are specific for cells expressing such polypeptides. The disclosed compositions are useful, for example, in the diagnosis, prevention and/or treatment of diseases, particularly colon cancer.

L5 ANSWER 40 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2002:165204 USPATFULL
TITLE: Cocoa extract compounds and methods for making and using the same
INVENTOR(S): Romanczyk, Leo J., JR., Hackettstown, NJ, UNITED STATES
Hammerstone, John F., JR., Nazareth, PA, UNITED STATES
Buck, Margaret M., Morristown, NJ, UNITED STATES
Post, Laurie S., Great Meadows, NJ, UNITED STATES
Cipolla, Giovanni G., Alpha, NJ, UNITED STATES
McClelland, Craig A., East Stroudsburg, PA, UNITED STATES
Mundt, Jeff A., Hackettstown, NJ, UNITED STATES
Schmitz, Harold H., Branchburg, NJ, UNITED STATES
PATENT ASSIGNEE(S): Mars, Incorporated (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002086833	A1	20020704
	US 6638971	B2	20031028
APPLICATION INFO.:	US 2001-776649	A1	20010205 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1997-831245, filed on 2 Apr 1997, GRANTED, Pat. No. US 6297273 Continuation-in-part of Ser. No. US 1996-631661, filed on 2 Apr 1996, ABANDONED
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Clifford Chance Rogers & Wells LLP, 200 Park Avenue, New York, NY, 10166-0153
NUMBER OF CLAIMS: 208
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 240 Drawing Page(s)
LINE COUNT: 5797
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Polyphenol-containing compositions, for example cocoa procyanidin monomer and/or oligomer-containing compositions, and their use for inhibiting bacterial growth are disclosed. Compositions may be used for

human and veterinary animal administration and may be, for example, in a
form of a food, a dietary supplement, or a pharmaceutical.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 41 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2002:181713 USPATFULL
TITLE: Cocoa extract compounds and methods for making and
using the same
INVENTOR(S): Romancyzk, Jr., Leo J., Hackettstown, NJ, United States
PATENT ASSIGNEE(S): Mars Incorporated, McLean, VA, United States (U.S.
corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6423743	B1	20020723
APPLICATION INFO.:	US 2000-717833		20001121 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1997-831245, filed
on 2 Apr 1997, now patented, Pat. No. US 6297273
Continuation-in-part of Ser. No. US 1996-631661, filed
on 2 Apr 1996, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Solola, T. A.
LEGAL REPRESENTATIVE: Kelley, Margaret B., Clifford Chance Rogers & Wells
NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 246 Drawing Figure(s); 234 Drawing Page(s)
LINE COUNT: 4656
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed and claimed are cocoa extracts, compounds, combinations
thereof and compositions containing the same, such as polyphenols or
procyanidins, methods for preparing such extracts, compounds and
compositions, as well as uses for them, especially a polymeric compound
of the formula A.sub.n, wherein A is a monomer of the formula: ##STR1##

wherein

n is an integer from 2 to 18, such that there is at least one terminal
monomeric unit A, and one or a plurality of additional monomeric units;

R is 3-(.alpha.)-OH, 3-(.beta.)-OH, 3-(.alpha.)-O-sugar, or
3-(.beta.)-O-sugar.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 42 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 6
ACCESSION NUMBER: 2002:239413 CAPLUS
DOCUMENT NUMBER: 136:354082
TITLE: Uptake and processing of Chlamydia trachomatis by
human dendritic cells
AUTHOR(S): Matyszak, Malgosia K.; Young, Joyce L.; Gaston, J. S.
Hill
CORPORATE SOURCE: Department of Medicine, Addenbrooke's Hospital,
University of Cambridge Clinical School, Cambridge, UK
SOURCE: European Journal of Immunology (2002), 32(3), 742-751
CODEN: EJIMAF; ISSN: 0014-2980
PUBLISHER: Wiley-VCH Verlag GmbH
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Chlamydia trachomatis (CT) causes several sexually transmitted diseases.
In 2-5% of cases, CT infection leads to the development of reactive
arthritis. Dendritic cells (DC) are central in T cell priming and the
induction of antigen specific immunity. Here the authors have studied the
uptake and processing of CT serovar L2 by human DC, and their ability to
present CT antigens to both CD4+ and CD8+ T cells. The authors show that
the entry of CT was mediated by the attachment of CT to heparan sulfates
and could be inhibited by heparin. There was ***no***
inhibition of uptake by an agent which blocks micropinocytosis.
Infecting DC with CT led to their activation and the prodn. of ***IL***
- ***12*** and TNF-.alpha. but not IL-10. Following invasion, CT was
confined to distinct vacuoles which were visualized with anti-CT
antibodies using confocal microscopy. Unlike with epithelial cells, these
vacuoles did not develop into characteristic inclusion bodies. In the
first 48 h, CT+ vacuoles were neg. for Lamp-1 and MHC class II. Despite
no obvious co-localization between CT vacuoles and MHC loading
compartments, infected DC efficiently presented CT antigens to CD4+ T

cells. Infected DC also expanded CT specific CD8+ T cells, allowing the
authors to generate a no. of CT-reactive CD8+ T cell clones. There is
still controversy about the importance of chlamydia-specific CD8+ T cell
responses in patients with arthritis. This is largely due to the
difficulties in studying CTL responses at the clonal level. The use of DC
as antigen-presenting cells should enable more detailed characterization
of these CTL responses.
REFERENCE COUNT: 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 43 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 7
ACCESSION NUMBER: 2003:78466 CAPLUS
DOCUMENT NUMBER: 139:159652
TITLE: In vitro effects of cAMP-elevating agents and
glucocorticoid either alone or in combination on the
production of nitric oxide, ***interleukin*** -
12 and interleukin-10 in IFN-.gamma.- and
LPS-activated mouse peritoneal macrophages
AUTHOR(S): Mittal, J.; Dogra, N.; Dass, R.; Majumdar, S.
CORPORATE SOURCE: Institute of Microbial Technology, Council of
Scientific and Industrial Research, Chandigarh, 160
036, India
SOURCE: Folia Microbiologica (Prague, Czech Republic) (2002),
47(6), 709-716
CODEN: FOMIAZ; ISSN: 0015-5632
PUBLISHER: Institute of Microbiology, Academy of Sciences of the
Czech Republic
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effects of cAMP-elevating agents, N6-2'-O-dibutyryl cAMP (Bu2cAMP),
and glucocorticoid (dexamethasone) on the prodn. of inflammatory mediators
- nitric oxide and ***interleukin*** - ***12*** (***IL*** -
12) and anti-inflammatory mediator interleukin-10 (IL-10) were
demonstrated in murine peritoneal macrophages. Inducible nitric oxide
synthase (iNOS) and iNOS mRNA were detected by northern blot and western
blot, resp. The cAMP elevating agents Bu2cAMP and prostaglandin E2 each
alone did not show any effect on NO prodn. but along with IFN-.gamma. and
lipopolysaccharide (LPS) they slightly enhanced NO prodn. Dexamethasone
inhibited NO prodn. in IFN-.gamma.- and LPS-treated cells; cAMP elevating
agents interfered with the NO prodn. inhibited by dexamethasone.
Inhibition was revealed at the mRNA level as well as at protein level.
Bu2cAMP or dexamethasone either alone or synergistically inhibited
IL - ***12*** prodn.; Bu2cAMP interfered with
dexamethasone-mediated inhibition of IL-10 prodn. in IFN-.gamma.- and
LPS-treated macrophages. The use of glucocorticoids along with cAMP
elevating agents was beneficial in lowering the level of inflammatory
mediator ***IL*** - ***12*** and producing high levels of the
anti-inflammatory mediator IL-10 active in cell protection. On the other
hand, interference of Bu2cAMP with dexamethasone-mediated ***NO***
inhibition may have adverse effect. Therefore, adverse effects
due to cAMP-mediated interference (inhibition) with NO synthesis may occur
in many inflammatory diseases during combined drug therapy by
glucocorticoids and cAMP elevating agents.
REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 44 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2002:744100 CAPLUS
DOCUMENT NUMBER: 138:121545
TITLE: Effects of tumor supernatant of A549 lung
adenocarcinoma on human monocyte-derived dendritic
cells
AUTHOR(S): Chen, Xiaobing; Zhao, Mingyao; Yang, Hongyan; Huang,
Youtian; Zheng, Zhimin; Ma, Junfen; Tang, Liming;
Dong, Ziming
CORPORATE SOURCE: Department of Pathophysiology, Basic Medical College,
Zhengzhou University, Zhengzhou, 450052, Peop. Rep.
China
SOURCE: Zhengzhou Daxue Xuebao, Yixueban (2002), 37(2),
139-143
CODEN: ZDXYBA; ISSN: 1671-6825

PUBLISHER: Zhengzhou Daxue Xuebao, Yixueban Bianjibu
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB Aim: To investigate the effects of tumor supernatant (TSN) of A549 lung adenocarcinoma on human monocyte-derived dendritic cells (MoDC), and to study whether functional MoDC pulsed with A549 tumor antigen is effective in inducing antitumor immune response. Methods: Human TSN, inactivated TSN and tumor antigen were prepd. The 2 kinds of TSN were added to the MoDC culture medium throughout the whole culture course or only at the late stage (the 7th d) in 7 groups with different culture conditions; MoDC were pulsed with tumor antigen on the 4th d or not and were collected on the 9th d. MoDC phenotypes were analyzed by flow cytometry (FCM); the cell apoptosis was obsd. by PI staining and FCM. Function was evaluated by mixed lymphocyte reaction (MLR), cytotoxic T lymphocyte (CTL) assay and ***IL*** - ***12*** in the supernatant was detected by using ELISA. Results: When MoDC exposed to TSN in the whole culture course, phenotypes showed unobviously, the apoptotic cell ratio was significantly higher, MLR, CTL, and ***IL*** - ***12*** decreased significantly. When MoDC exposed to TSN at the late culture stage or exposed to inactivated TSN throughout the whole culture course, ***no*** ***inhibition*** effects as above were demonstrated. When MoDC were free from TSN and pulsed with lung adenocarcinoma cell antigen, the phenotypes and function showed obviously. Conclusions: TSN could up-regulate phenotypic maturation of MoDC, induce apoptosis and inhibit the antitumor immunity of mature MoDC. The normal MoDC pulsed with tumor antigen could induce higher antitumor immune response in vitro.

L5 ANSWER 45 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2002:421849 CAPLUS
DOCUMENT NUMBER: 137:80
TITLE: Immunopotentiating activity of nigerooligosaccharides
AUTHOR(S): Yamamoto, Yoshihiro
CORPORATE SOURCE: Research and Development Section, Takeda Food Products, Ltd., Itami, 664-0011, Japan
SOURCE: Fragrance Journal (2002), 30(5), 50-58
CODEN: FUJAD7; ISSN: 0288-9803
PUBLISHER: Fureguransu Janaru Sha
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese
AB A review on the enhancement of ***IL*** - ***12*** and IFN-.gamma. formation by nigerooligosaccharides (NOS; nigerose, nigerosylglucose, etc.), augmentation of NK activity by NOS, and immunopotentiating effects of NOS in murine disease models and humans. Mice fed a NOS diet showed longer survival time after the induction of endogenous infection, and ***NOS*** ***inhibited*** tumor cell proliferation in mice. Daily dietary intake of NOS augmented immune functions in healthy humans and improved health-related QOL in the healthy elderly humans.

L5 ANSWER 46 OF 74 USPATFULL on STN
ACCESSION NUMBER: 2001:168156 USPATFULL
TITLE: Use of cocoa solids having high cocoa polyphenol content in tabletting compositions and capsule filling compositions
INVENTOR(S): Romanczyk, Jr., Leo J., Hackettstown, NJ, United States
PATENT ASSIGNEE(S): Mars, Inc., McLean, VA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6297273	B1	20011002
APPLICATION INFO.:	US 1997-831245		19970402 (8)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Tsang, Cecilia
ASSISTANT EXAMINER: Solola, Taofiq A.
LEGAL REPRESENTATIVE: Kelley, Margaret B.Clifford Chance Rogers & Wells, LLP
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 237 Drawing Figure(s); 221 Drawing Page(s)
LINE COUNT: 4861
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed and claimed are cocoa extracts, compounds, combinations thereof and compositions containing the same, such as polyphenols or procyanidins, methods for preparing such extracts, compounds and compositions, as well as uses for them, especially a polymeric compound

of the formula A.sub.n, wherein A is a monomer of the formula: ##STR1##

wherein n is an integer from 2 to 18, such that there is at least one terminal monomeric unit A, and one or a plurality of additional monomeric units;

R is 3-(.alpha.)-OH, 3-(.beta.)-OH, 3-(.alpha.)-O-sugar, or 3-(.beta.)-O-sugar;

bonding between adjacent monomers takes place at positions 4, 6 or 8;

a bond of an additional monomeric unit in position 4 has alpha or beta stereochemistry;

X, Y and Z are selected from the group consisting of monomeric unit A, hydrogen, and a sugar, with the provisos that as to the at least one terminal monomeric unit, bonding of the additional monomeric unit thereto (the bonding of the additional monomeric unit adjacent to the terminal monomeric unit) is at position 4 and optionally Y=Z=hydrogen;

the sugar is optionally substituted with a phenolic moiety, at any position on the sugar, for instance via an ester bond, and

pharmaceutically acceptable salts or derivatives thereof (including oxidation products).

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 47 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 8
ACCESSION NUMBER: 2001:867444 CAPLUS
DOCUMENT NUMBER: 136:131323
TITLE: Host response to infection: the role of CpG DNA in induction of cyclooxygenase 2 and nitric oxide synthase 2 in murine macrophages
AUTHOR(S): Ghosh, Dipak K.; Misukonis, Mary A.; Reich, Charles; Pisetsky, David S.; Weinberg, J. Brice
CORPORATE SOURCE: Department of Medicine, Veterans Affairs and Duke University Medical Centers, Durham, NC, 27705, USA
SOURCE: Infection and Immunity (2001), 69(12), 7703-7710
CODEN: INFIBR; ISSN: 0019-9567
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Depending on sequence, bacterial and synthetic DNAs can activate the host immune system and influence the host response to infection. The purpose of this study was to det. the abilities of various phosphorothioate oligonucleotides with cytosine-guanosine-contg. motifs (CpG DNA) to activate macrophages to produce nitric oxide (NO) and prostaglandin E2 (PGE2) and to induce expression of NO synthase 2 (NOS2) and cyclooxygenase 2 (COX2). As little as 0.3 .mu.g of CpG DNA/mL increased NO and PGE2 prodn. in a dose- and time-dependent fashion in cells of the mouse macrophage cell line J774. NO and PGE2 prodn. was noted by 4 to 8 h after initiation of cultures with the CpG DNA, with the kinetics of NO prodn. induced by CpG DNA being comparable to that induced by a combination of lipopolysaccharide and gamma interferon. CpG DNA-treated J774 cells showed enhanced expression of NOS2 and COX2 proteins as detd. by immunoblotting, with the relative potencies of the CpG DNAs generally corresponding to those noted for the induction of NO and PGE2 prodn. as well as to those noted for the induction of interleukin-6 (IL-6), ***IL*** - ***12*** , and tumor necrosis factor. Exts. from CpG DNA-treated cells converted L-arginine to L-citrulline, but the ***NOS*** ***inhibitor*** NG-monomethyl-L-arginine (NMMA) inhibited this reaction. The COX2-specific inhibitor NS398 inhibited CpG DNA-induced PGE2 prodn. and inhibited NO prodn. to various degrees. The ***NOS*** ***inhibitors*** NMMA, 1400W, and N-iminoethyl-L-lysine effectively blocked NO prodn. and increased the prodn. of PGE2 in a dose-dependent fashion. Thus, analogs of microbial DNA (i.e., CpG DNA) activate mouse macrophage lineage cells for the expression of NOS2 and COX2, with the prodn. of NO and that of PGE2 occurring in an interdependent manner.
REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 48 OF 74 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
ACCESSION NUMBER: 2001241683 EMBASE
TITLE: Natural killer cells and nitric oxide.
AUTHOR: Cifone M.G.; Ulisse S.; Santoni A.
CORPORATE SOURCE: M.G. Cifone, Department of Experimental Medicine,
University of L'Aquila, Via Vetoio, 10 Coppito 2, 67100
L'Aquila, Italy. cifone@univaq.it
SOURCE: International Immunopharmacology, (2001) 1/8 (1513-1524).
Refs: 103
ISSN: 1567-5769 CODEN: IINMBA
PUBLISHER IDENT.: S 1567-5769(01)00095-9
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 026 Immunology, Serology and Transplantation
030 Pharmacology
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Natural killer (NK) cells and nitric oxide (NO) are both important
components of the natural or innate immune response. NK cells are large
granular lymphocytes capable of destroying cells infected by virus or
bacteria and susceptible tumor cells without prior sensitization and
restriction by MHC antigens. They are abundant in blood, spleen, liver and
lungs and are distinct from both T and B lymphocytes in their circulation
patterns, profile of surface antigens, receptor repertoire and the way in
which they discriminate between self and non-self. Uniquely, NK cells
express receptors that can recognize and discriminate between normal and
altered MHC class I determinants. NK cell cytotoxic activity is strongly
induced by cytokines such as IL-2 and ***IL*** - ***12*** , and this
activation is associated with synthesis of ***NO*** .
Inhibitors of NO synthesis impair NK cell-mediated target cell
killing, demonstrating a role for NO in NK cell function. Furthermore, NO
itself can regulate NK cell activation. In this article, evidence that NO
is a mediator of NK cell-mediated target cell killing, and that NO is a
regulator of NK cell activation will be reviewed. Results of NO synthase
gene deletion studies will be discussed, and rodent and human NK cells
will be compared. .COPYRGT. 2001 Elsevier Science B.V.

L5 ANSWER 49 OF 74 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN
ACCESSION NUMBER: 2001:258354 BIOSIS
DOCUMENT NUMBER: PREV200100258354
TITLE: Inhibition of ***interleukin*** - ***12*** (
IL - ***12***) p40 transcription by nitric oxide
(NO) in murine macrophages.
AUTHOR(S): Xiong, Huabao [Reprint author]; Zhu, Chen [Reprint author];
Plevy, Scott E. [Reprint author]
CORPORATE SOURCE: Mount Sinai School of Medicine, One Gustave L. Levy Place,
New York, NY, 10029, USA
SOURCE: FASEB Journal, (March 8, 2001) Vol. 15, No. 5, pp. A1037.
print.
Meeting Info.: Annual Meeting of the Federation of American
Societies for Experimental Biology on Experimental Biology
2001. Orlando, Florida, USA. March 31-April 04, 2001.
CODEN: FAJOEC. ISSN: 0892-6638.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 30 May 2001
Last Updated on STN: 19 Feb 2002
AB Macrophage-derived NO is an important effector molecule of the innate
immune system. During an immune response, NO may influence adaptive
immunity. Therefore, we studied whether macrophage and dendritic cell
derived NO can alter the expression of ***IL*** - ***12*** . In a
dose-dependent manner, S-nitroso-N-acetylpenicillamine (SNAP), an NO
donor, inhibited LPS-induced ***IL*** - ***12*** p40 and p35 mRNA
expression (RNase protection assay) and ***IL*** - ***12*** p40
protein production (ELISA) in bone marrow derived murine macrophages,
dendritic cells, and the murine macrophage cell line RAW 264.7. In a RAW
264.7 derivative that does not produce NO, increased ***IL*** -
12 p40 mRNA was detected. The ***IL*** - ***12*** p40
promoter is regulated through two important cis-acting control elements
that bind NF-kB and C/EBP family members. SNAP inhibited LPS-induced
IL - ***12*** p40 promoter activity in RAW264.7 cells as
determined by luciferase assay. This strong inhibitory effect was also

detected using a minimal promoter from -101 to +55 that contains the C/EBP and downstream elements, but not the NF-kB site. In nuclear extracts from LPS-activated RAW 264.7 cells, electrophoretic mobility shift assays revealed that SNAP pretreatment strongly reduced C/EBP and NF-kB DNA binding to the p40 promoter. As IL-10 can potently inhibit ***IL*** - ***12*** , the effects of NO on IL-10 expression was studied. ***NO*** ***inhibited*** IL-10 mRNA accumulation and promoter activity in RAW 264.7 cells. In IL-10 deficient mice, NO strongly inhibited ***IL*** - ***12*** protein production from macrophages, demonstrating that the inhibitory effects of NO are independent of IL-10. In summary, these results indicate that NO is a potent inhibitor of ***IL*** - ***12*** p40 gene expression in macrophages and dendritic cells. NO may inhibit ***IL*** - ***12*** p40 transcription by attenuating NF-kB and C/EBP activation. These experiments provide another example of how an innate immune effector may have a profound effect on adaptive immunity.

L5 ANSWER 50 OF 74 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2001369454 EMBASE
TITLE: Nitric oxide and the immune response.
AUTHOR: Bogdan C.
CORPORATE SOURCE: C. Bogdan, Institute of Clinical Microbiology, F.-A.-Univ. Erlangen-Nuremberg, Wasserturmstrasse 3-5, D-91054 Erlangen, Germany. christian.bogdan@mikrobio.med.uni-erlangen.de
SOURCE: Nature Immunology, (2001) 2/10 (907-916). Refs: 189 ISSN: 1529-2908 CODEN: NIAMCZ
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
016 Cancer
026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB During the past two decades, nitric oxide (NO) has been recognized as one of the most versatile players in the immune system. It is involved in the pathogenesis and control of infectious diseases, tumors, autoimmune processes and chronic degenerative diseases. Because of its variety of reaction partners (DNA, proteins, low-molecular weight thiols, prosthetic groups, reactive oxygen intermediates), its widespread production (by three different NO synthases (NOS) and the fact that its activity is strongly influenced by its concentration, NO continues to surprise and perplex immunologists. Today, there is no simple, uniform picture of the function of NO in the immune system. Protective and toxic effects of NO are frequently seen in parallel. Its striking inter- and intracellular signaling capacity makes it extremely difficult to predict the effect of ***NOS*** ***inhibitors*** and NO donors, which still hampers therapeutic applications.

L5 ANSWER 51 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 9
ACCESSION NUMBER: 2001:598559 CAPLUS
DOCUMENT NUMBER: 135:287309
TITLE: Effects of prostaglandin E2 and nitric oxide inhibitors on the expression of interleukin-10, ***interleukin*** - ***12*** and MHC class-II molecules in Mycobacterium microti-infected and interferon-.gamma.-treated mouse peritoneal macrophages
AUTHOR(S): Mittal, J.; Dogra, N.; Vohra, H.; Majumdar, S.
CORPORATE SOURCE: Institute of Microbial Technology, Chandigarh, 160 036, India
SOURCE: Folia Microbiologica (Prague, Czech Republic) (2001), 46(3), 259-264 CODEN: FOMIAZ; ISSN: 0015-5632
PUBLISHER: Institute of Microbiology, Academy of Sciences of the Czech Republic
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Mycobacterium microti-infected mouse peritoneal macrophages produced high amts. of prostaglandin E2 (PGE2) and nitric oxide (NO) when activated with interferon-.gamma. (IFN-.gamma.). In order to understand the relation

between PGE2 and NO prodn. and the expression of ***interleukin*** - ***12*** (***IL*** - ***12***), interleukin-10 (IL-10) and MHC class-II (Ia) mols. by M. microti-infected and IFN-.gamma.-stimulated macrophages, we analyzed the level of these mols. in the presence or absence of PGE2 and ***NO*** ***inhibitors*** . Addn. of NG-methyl-L-arginine (L-NMA) and indomethacin (IM) caused a significant increase in ***IL*** - ***12*** level (2.6- and 1.9-fold, resp.) whereas IL-10 level decreased by 88 and 56%, resp., relative to M. microti-infected and IFN-.gamma.-treated control macrophages. Enhanced PGE2 and NO upregulated IL-10 expression and down-regulated ***IL*** - ***12*** and MHC class-II (Ia) expression in M. microti-infected and IFN-.gamma.-treated mouse peritoneal macrophages.
REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 52 OF 74 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2002182701 EMBASE
TITLE: Angiogenesis: From the molecular mechanisms to the development of new drugs.
AUTHOR: Morbidelli L.; Donnini S.; D'Amore V.; Ziche M.
CORPORATE SOURCE: L. Morbidelli, Istituto di Scienze Farmacologiche, Universita di Siena, Siena, Italy
SOURCE: Acta Medica Romana, (2001) 39/2 (238-246).
Refs: 24
ISSN: 0001-6098 CODEN: AMROBA
COUNTRY: Italy
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English; Italian
AB The steps required for new vessel growth are biologically complex and require coordinate regulation of contributing components, including modifications of cell-cell interactions, proliferation and migration of endothelial cells and matrix degradation. The observation that in vivo angiogenesis is accompanied by vasodilation, that many angiogenesis effectors possess vasodilating properties and that tumor vasculature is in a persistent state of vasodilation, support the existence of a molecular/biochemical link between vasodilation and angiogenesis. Several pieces of evidence converge in the indication of a role for nitric oxide (NO), the factor responsible for vasodilation, in physiological and pathological angiogenesis. Data originated in different labs indicate that NO can act both as an "actor" of angiogenesis and as a "director of angiogenesis", both functions being equally expressed during physiological and pathological processes. NO significantly contributes to the prosurvival/proangiogenic program of capillary endothelium by triggering and transducing cell growth and differentiation via endothelial-constitutive NO synthase (ec-NOS) activation, cyclic GMP (cGMP) elevation, mitogen activated kinase (MAPK) activation and fibroblast growth factor-2 (FGF-2) expression. Re-establishment of a balanced NO production in the cardiovascular system results in a reduction of cell damage during inflammatory and vascular diseases. Elevation of NOS activity in correlation with angiogenesis and tumor progression has been extensively reported in experimental and human tumors. Tumor expansion and edema formation are sensitive to ***NOS*** ***inhibition*** . On this basis, the nitric oxide pathway appears to be a promising target for consideration in pro- and antiangiogenic therapeutic strategies. The use of ***NOS*** ***inhibitors*** seems appropriate to reduce edema, block angiogenesis and facilitate antitumor drug delivery.

L5 ANSWER 53 OF 74 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 2001:246868 BIOSIS
DOCUMENT NUMBER: PREV200100246868
TITLE: Modulation of nitric oxide synthase and cyclooxygenase 2 by CpG DNA in murine macrophages.
AUTHOR(S): Ghosh, Dipak K. [Reprint author]; Misukonis, Mary [Reprint author]; Mast, Molly [Reprint author]; Reigh, Charles

[Reprint author]; Pisetsky, David [Reprint author];
Weinberg, Joe Brice [Reprint author]
CORPORATE SOURCE: Medicine/Hem-oncology, Duke university and VA Medical
center, 508 fulton street, Durham, NC, 27705, USA
SOURCE: FASEB Journal, (March 7, 2001) Vol. 15, No. 4, pp. A200.
print.
Meeting Info.: Annual Meeting of the Federation of American
Societies for Experimental Biology on Experimental Biology
2001. Orlando, Florida, USA. March 31-April 04, 2001.
CODEN: FAJOEC. ISSN: 0892-6638.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 23 May 2001
Last Updated on STN: 19 Feb 2002
AB Bacterial and synthetic DNA can act an immune stimulators and induce
inflammation in vivo. The purpose of this study was to determine the
abilities of various phosporothioated, cytosine guanosine-containing DNAs
("CpG-DNA") to activate mouse macrophages for nitric oxide (NO) and
prostaglandin E2 (PGE2) production and inducible NO synthase (NOS2) and
cyclooxygenase (COX2) expression. As little as 0.3 ug/ml CpG-DNA
increased NO and PGE2 production in a dose- and time-dependent fashion.
An oligonucleotide containing 2 CpG sequences ("SAK2") was generally the
most potent. NO and PGE2 production was noted by 4 to 8 hours after
initiation of cultures with CpG-DNA, with kinetics of NO production for
CpG-DNA being comparable to that induced by LPS/IFN-gamma. LPS,
IFN-gamma., or LPS/IFN-gamma did not induce PGE2 production. J774 cells
treated with CpG-DNA had enhanced expression of NOS2 and COX2 protein as
determined by immunoblot, with the relative potencies of the DNAs
corresponding to that noted for induc-tion of NO and PGE2 production, as
well as that for induction of IL-6, ***IL*** - ***12*** , and TNF.
Extracts from CpG-DNA-treated cells converted L-arginine to L-citrulline,
and this was inhibited by the ***NOS*** ***inhibitor*** NMMA. The
COX2-specific inhibitor NS398 completely inhibited CpG-DNA-induced PGE2
production, but had no effect on NO production. The ***NOS***
inhibitors NMMA, 1400W, and L-NIL effectively blocked NO
production, but increased production of PGE2 in a dose-dependent fashion.
Thus, CpG-DNA activates mouse macrophages for expression of NOS2 and COX2
and production of the pro-inflammatory mediators NO and PGE2.

L5 ANSWER 54 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 10
ACCESSION NUMBER: 2000:466559 CAPLUS
DOCUMENT NUMBER: 133:191684
TITLE: ***Interleukin*** - ***12*** (***IL*** -
12) enhancement of the cellular immune
response against human immunodeficiency virus type 1
env antigen in a DNA prime/vaccinia virus boost
vaccine regimen is time and dose dependent:
suppressive effects of ***IL*** - ***12*** boost
are mediated by nitric oxide
AUTHOR(S): Gherardi, M. Magdalena; Ramirez, Juan C.; Esteban,
Mariano
CORPORATE SOURCE: Department of Molecular and Cellular Biology, Centro
Nacional de Biotecnologia, CSIC, Universidad Autonoma,
Madrid, E-28049, Spain
SOURCE: Journal of Virology (2000), 74(14), 6278-6286
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The authors previously demonstrated that codelivery of ***interleukin***
- ***12*** (***IL*** - ***12***) with the human immunodeficiency
virus type 1 (HIV-1) Env antigen from a recombinant vaccinia virus (rVV)
can enhance the specific anti-Env cell-mediated immune (CMI) response.
Here, they investigated the effects of ***IL*** - ***12*** in mice
when it is expressed in a DNA prime/VV boost vaccine regimen. The
delivery of ***IL*** - ***12*** and Env product during priming with
a DNA vector, followed by a booster with VV expressing the Env gene
(rVVenv), was found to trigger the optimal CMI response compared with
other immunization schedules studied. Significantly, if ***IL*** -

12 is also delivered as a booster from the viral vector, an
impairment of the effects of ***IL*** - ***12*** was obsd. involving
nitric oxide (NO), since it was overcome by specific inhibitors of
inducible NO synthase. NO caused transient immunosuppression rather than
impairment of viral replication. Moreover, at certain viral doses,
coadministration of the ***NO*** ***inhibitor*** during the
booster resulted in ***IL*** - ***12*** - mediated enhancement of the
specific CD8+ T-cell response. In addn., the dose of the ***IL*** -
12 -encoding plasmid (pIL-12) and the route of administration of
both vectors were relevant factors for optimal CMI responses. Maximal
nos. of Env-specific CD8+ .gamma. interferon-secreting cells were obtained
when 50 .mu.g of pIL-12 was administered i.m. at priming, followed by an
i.v. rVVenv boost. The authors' results demonstrate, in a murine model,
crit. parameters affecting the success of vaccination schedules based on a
combination of DNA and VV vectors in conjunction with immunomodulators.
REFERENCE COUNT: 59 THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 55 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 11
ACCESSION NUMBER: 2000:603722 CAPLUS
DOCUMENT NUMBER: 133:280428
TITLE: Babesia bovis-stimulated macrophages express
interleukin-1.beta., ***interleukin*** - ***12***
, tumor necrosis factor alpha, and nitric oxide and
inhibit parasite replication in vitro
AUTHOR(S): Shoda, Lisl K. M.; Palmer, Guy H.; Florin-Christensen,
Jorge; Florin-Christensen, Monica; Godson, Dale L.;
Brown, Wendy C.
CORPORATE SOURCE: Program in Vector-Brone Diseases, Department of
Veterinary Microbiology and Pathology, Washington
State University, Pullman, WA, 99164-7040, USA
SOURCE: Infection and Immunity (2000), 68(9), 5139-5145
CODEN: INFIBR; ISSN: 0019-9567
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The tick-transmitted hemoparasite Babesia bovis causes an acute infection
that results in persistence and immunity against challenge infection in
cattle that control the initial parasitemia. Resoln. of acute infection
with this protozoal pathogen is believed to be dependent on products of
activated macrophages (M.PHI.), including inflammatory cytokines and
nitric oxide (NO) and its derivs. B. bovis stimulates inducible nitric
oxide synthase (iNOS) and prodn. of NO in bovine M.PHI., and chem. donors
of. ***NO*** ***inhibit*** the growth of B. bovis in vitro.
However, the induction of inflammatory cytokines in M.PHI. by babesial
parasites has not been described, and the antiparasitic activity of NO
produced by B. bovis-stimulated M.PHI. has not been definitively
demonstrated. We report that monocyte-derived M.PHI. activated by B.
bovis expressed enhanced levels of inflammatory cytokines
interleukin-1.beta. (IL-1.beta.), ***IL*** - ***12*** , and tumor
necrosis factor alpha that are important for stimulating innate and
acquired immunity against protozoal pathogens. Furthermore, a lipid
fraction of B. bovis-infected erythrocytes stimulated iNOS expression and
NO prodn. by M.PHI.. Cocultures of M.PHI. and B. bovis-infected
erythrocytes either in contact or phys. sepd. resulted in reduced parasite
viability. However, NO produced by bovine M.PHI. in response to B.
bovis-infected erythrocytes was only partially responsible for parasite
growth inhibition, suggesting that addnl. factors contribute to the
inhibition of B. bovis replication. These findings demonstrate that B.
bovis induces an innate immune response that is capable of controlling
parasite replication and that could potentially result in host survival
and parasite persistence.
REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 56 OF 74 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 12

ACCESSION NUMBER: 2000249268 EMBASE
TITLE: Targeting activated lymphocytes with an entirely human
immunotoxin analogue: Human pancreatic RNase1-human
IL - ***12*** fusion.
AUTHOR: Psarras K.; Ueda M.; Tanabe M.; Kitajima M.; Aiso S.;
Komatsu S.; Seno M.
CORPORATE SOURCE: Dr. M. Ueda, Department of Surgery, Keio University School
of Medicine, 35 Shinanomachi, Tokyo 160-8582, Japan
SOURCE: Cytokine, (2000) 12/6 (786-790).
Refs: 15
ISSN: 1043-4666 CODEN: CYTIE
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
AB A hybrid human protein was produced in E. coli by fusing the genes
encoding human pancreatic RNase1 (hpRNase1) and human IL-2 (hIL-2). The
recombinant hpRNase1-hIL-2 inhibited protein synthesis in HTLV-1-infected,
malignant T cells, which hyperproduce high affinity IL-2 receptors, with
an IC50 of 2 x 10-8 M, whereas ***no*** ***inhibition*** was
detectable in control cells with lower affinity receptors. HpRNase1 alone
had an IC50 of almost 10-3 M. A molar excess of hIL-2 blocked the protein
synthesis inhibition dose-dependently. In a human mixed lymphocyte
culture, hpRNase1-hIL-2 inhibited the proliferation of responder cells
with potency comparable to that of cyclosporine, while non-effective doses
of FK506 importantly improved its potency. Despite its short half-life in
animals, hpRNase1-hIL-2 rapidly enters cells in a few minutes and arrests
the protein translation in less than 10 h. Thus, hpRNase1-hIL-2 may be
useful to selectively eliminate activated lymphocytes hyperproducing high
affinity IL-2 receptors, as in allograft rejection, gaft-versus-host
disease, autoimmune disorders, adult T cell leukaemia and other
lymphoproliferative or retroviral malignancies including HIV infection,
without inducing general immunosuppression. As an entirely human
'immunotoxin analogue' it may alleviate the dose limiting toxicity and
immunogenicity of conventional immunotoxins.
(C) 2000 Academic Press.

L5 ANSWER 57 OF 74 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 13
ACCESSION NUMBER: 2000:428915 BIOSIS
DOCUMENT NUMBER: PREV200000428915
TITLE: ***Interleukin*** - ***12*** enhances the antitumor
activity of cytotoxic T lymphocytes against lung
adenocarcinoma engrafted in severe combined immunodeficient
mice.
AUTHOR(S): Hanagiri, Takeshi [Reprint author]; Imahayashi, Satoru;
Yoshino, Ichiro; So, Tomoko; Eifuku, Ryozo; Yoshimatsu,
Takashi; Takenoyama, Mitsuhiro; Osaki, Toshihiro;
Nakanishi, Ryoich; Ichiyoshi, Yuji; Nomoto, Kikuo;
Yasumoto, Kosei
CORPORATE SOURCE: Second Department of Surgery, School of Medicine,
University of Occupational and Environmental Health, 1-1
Iseigaoka, Yahatanishi-ku, Kitakyushu, 807-8555, Japan
SOURCE: International Journal of Clinical Oncology, (August, 2000)
Vol. 5, No. 4, pp. 262-268. print.
ISSN: 1341-9625.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Oct 2000
Last Updated on STN: 10 Jan 2002
AB Background: Through a number of biologic activities, ***interleukin***
12 (***IL*** - ***12***) has proven to be a potential
antitumor cytokine in mice bearing a variety of malignancies. However, in
clinical trials in humans, the eradication of solid tumors remains
difficult. Methods: A lung cancer cell line (PC-9)-specific cytotoxic T
lymphocytes (CTL) were generated by multiple stimulations, with irradiated
PC-9 cells, of regional lymph node lymphocytes obtained from patients with
lung cancer whose cells expressed the same HLA-A locus haplotype as PC-9
(HLA-A24). Severe combined immunodeficient (SCID) mice bearing a
subcutaneous graft of PC-9 were then intravenously injected with
anti-PC-9-specific CTLs. Under these conditions, the in-vivo effect of
recombinant human (rh) IL-2 and rh ***IL*** - ***12*** was

evaluated, based on tumor growth. Results: Mice that received either rh
IL-2 or rh ***IL*** - ***12*** exhibited ***no***
inhibitory effect on tumor growth. However, mice that received
adoptive immunotherapy (AIT) alone exhibited a significant inhibition of
tumor growth in the PC-9 graft in comparison to untreated mice. When mice
were treated with AIT combined with rh IL-2 + rh ***IL*** - ***12***
administration, tumor growth was significantly suppressed. A significant
difference was observed in the growth of the PC-9 graft between AIT + IL-2
+ ***IL*** - ***12*** treatment and AIT + IL-2 treatment. Four of
eight mice in the AIT + IL-2 + ***IL*** - ***12*** -treated group
showed complete tumor regression. Conclusion: ***IL*** - ***12***
showed a synergistic effect with adoptive immunotherapy, using CTL in a
tumor-engrafted SCID model. These results are therefore considered to
provide a sufficient rationale for IL-2 + ***IL*** - ***12*** -based
immunotherapy using CTL transfer for patients with lung cancer.

L5 ANSWER 58 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 14
ACCESSION NUMBER: 2000:868538 CAPLUS
DOCUMENT NUMBER: 135:14036
TITLE: Differential anti-inflammatory and anti-oxidative
effects of dexamethasone and N-acetylcysteine in
endotoxin-induced lung inflammation
AUTHOR(S): Rocksen, D.; Lilliehook, B.; Larsson, R.; Johansson,
T.; Bucht, A.
CORPORATE SOURCE: Department of Biomedicine, Defence Research
Establishment, Umea, SE-90182, Swed.
SOURCE: Clinical and Experimental Immunology (2000), 122(2),
249-256
CODEN: CEXIAL; ISSN: 0009-9104
PUBLISHER: Blackwell Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Inhalation of bacterial endotoxin induces an acute inflammation in the
lower respiratory tract. In this study, the anti-inflammatory effects of
the anti-oxidant N-acetylcysteine (NAC) and the glucocorticoid
dexamethasone were investigated in mice exposed to aerosolized endotoxin
(lipopolysaccharide (LPS)). Powerful redn. of neutrophils in
bronchoalveolar lavage fluid (BALF) was obtained by a single i.p.
injection of dexamethasone (10 mg/kg), whereas treatment with NAC only
resulted in redn. of neutrophils when administered at a high dose (500
mg/kg). Measurement of cytokine and chemokine expression in lung tissue
revealed a decrease of tumor necrosis factor-alpha, IL-1.alpha.,
IL-1.beta., IL-6, IL-12p40, and MIP-1.alpha. mRNA when mice where treated
with dexamethasone but not when treated with NAC. Anal. of oxidative
burst demonstrated a remarkable redn. of oxygen radicals in BALF
neutrophils after treatment with dexamethasone, whereas the effect of NAC
was not different from that in untreated animals. In conclusion,
dexamethasone exerted both anti-inflammatory and anti-oxidative effects in
acute airway inflammation, probably by blocking early events in the
inflammatory cascade. In contrast, treatment with NAC resulted in a weak
redn. of the inflammatory response but ***no*** ***inhibition***
of pro-inflammatory cytokines or redn. of oxidative burst in neutrophils.
These results demonstrate dramatic differences in efficiency and also
indicate that the 2 drugs have different actions. Combined treatment with
NAC and dexamethasone revealed an additive action but no synergy was obsd.
REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 59 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 15
ACCESSION NUMBER: 2001:605707 CAPLUS
DOCUMENT NUMBER: 136:198507
TITLE: Phagocytosis of bacteria by mouse bone marrow-derived
dendritic cells affects their ability to process a
heterologous soluble antigen in vitro
AUTHOR(S): Bryniarski, Krzysztof; Biedron, Rafal; Petrovska,
Liliana; Free, Paul; Chain, Benjamin; Marcinkiewicz,
Janusz
CORPORATE SOURCE: Department of Immunology, Jagiellonian University
Medical College, Krakow, 31-121, Pol.

SOURCE: Central European Journal of Immunology (2000), 25(4), 210-215
CODEN: CJIMFW; ISSN: 1426-3912
PUBLISHER: Termedia
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Sentinel dendritic cells are likely to encounter both live and dead bacteria at sites of infection. Although dendritic cells can phagocytose such bacteria, their principle role is not in bacterial killing but in stimulation of a subsequent adaptive immune response. In contrast, neutrophils at the site of infection play a major role in bacterial killing, in part via oxidative chlorination by myeloperoxidase products. In this study, the interaction between bacterial phagocytosis and the antigen processing function of dendritic cells is examd. Ingestion of heat-killed Salmonella typhimurium, or bacteria killed by oxidative chlorination, induces up-regulation of co-stimulatory mols. on dendritic cells, and strong stimulation of the TH1-inducing proinflammatory cytokines ***IL*** - ***12*** and TNF-.alpha.. In contrast, induction of nitric oxide prodn. is weak. Finally, phagocytosis of bacteria inhibits processing of protein antigen, but only if phagocytosis precedes exposure to antigen by 24 h. Phagocytosis itself has ***no*** ***inhibitory*** effect on the concomitant processing and presentation of either protein or peptide antigen to T cells. These results demonstrate that both phagocytic and antigen processing pathways can operate simultaneously within dendritic cells, allowing these sentinel cells to operate effectively at the site of bacterial infection.
REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 60 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 16
ACCESSION NUMBER: 2000:102656 CAPLUS
DOCUMENT NUMBER: 132:249760
TITLE: Neutralizing antibodies to granulocyte-macrophage colony-stimulating factor, interleukin-1.alpha. and interferon-.alpha. but not other cytokines in human immunoglobulin preparations
AUTHOR(S): Wadhwa, M.; Meager, A.; Dilger, P.; Bird, C.; Dolman, C.; Das, R. G.; Thorpe, R.
CORPORATE SOURCE: Division of Immunobiology, National Institute for Biological Standards and Control, Potters Bar, EN6 3QG, UK
SOURCE: Immunology (2000), 99(1), 113-123
CODEN: IMMUAM; ISSN: 0019-2805
PUBLISHER: Blackwell Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Human Ig prepns. are used therapeutically for various disorders. Such therapy is generally safe but adverse effects occasionally occur in recipients. It has been suggested that antibodies to cytokines present in clin. Ig products may contribute to undesirable effects in recipients. Therefore, we investigated i.v. and i.m. Ig products for the presence of cytokine-specific neutralizing antibodies. Using validated bioassays, we detected neutralizing activity against human granulocyte-macrophage colony-stimulating factor (GM-CSF), interferon-.alpha.2a (IFN-.alpha.2a) and interleukin-1.alpha. (IL-1.alpha.) in Ig products. We found ***no*** ***neutralization*** of granulocyte colony-stimulating factor, macrophage colony-stimulating factor, stem cell factor, IL-1.beta., IL-2, IL-3, IL-4, IL-6, IL-9, IL-10, ***IL*** - ***12*** , tumor necrosis factor-.alpha., oncostatin M (OSM) and IFN-.gamma.. Most batches which neutralized IFN-.alpha.2a activity also neutralized other IFN-.alpha. subtypes, IFN-.omega. and IFN-.beta.. Most products (94%) neutralized the biol. activity of GM-CSF. No correlation between batches and their ability to neutralize bioactivities of GM-CSF, IFN-.alpha.2a and IL-1.alpha. was found. This neutralizing activity could be traced to plasma pools used for manuf. of Igs. The neutralization was mediated by specific cytokine antibodies contained within Ig products as it was present in specific IgG fractions eluted from cytokine affinity chromatog. columns. Specific binding of such IgG fractions to cytokines in immunoblots and in enzyme-linked immunosorbent assays (ELISAs) was obsd.

This contrasts with the broad non-specific recognition of cytokine
proteins obsd. using unfractionated Igs in ELISAs. This is the first
comprehensive study showing the presence of neutralizing antibodies
against GM-CSF, IL-1.alpha., or IFN-.alpha.2a in Ig products.
REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 61 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 17
ACCESSION NUMBER: 1999:510658 CAPLUS
DOCUMENT NUMBER: 131:125825
TITLE: Effects of nitric oxide on the induction and
differentiation of Th1 cells
AUTHOR(S): Niedbala, Wanda; Wei, Xiao-Qing; Piedrafita, David;
Xu, Damo; Liew, Foo Yew
CORPORATE SOURCE: Dep. Immunology, Univ. Glasgow, Glasgow, G11 6NT, UK
SOURCE: European Journal of Immunology (1999), 29(8),
2498-2505
CODEN: EJIMAF; ISSN: 0014-2980
PUBLISHER: Wiley-VCH Verlag GmbH
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The authors have previously shown that mice lacking inducible NO synthase
are markedly more susceptible to Leishmania major infection but developed
a significantly enhanced Th1 cell response compared with wild-type mice.
At high concns., ***NO*** ***inhibited*** ***IL*** - ***12***
synthesis by activated macrophages, thereby indirectly suppressing the
expansion of Th1 cells. We report that at low concns., NO selectively
enhanced the induction of Th1 cells and had no effect on Th2 cells. NO
exerted this effect in synergy with ***IL*** - ***12*** during Th1
cell differentiation and had no effect on fully committed Th1 cells. NO
appears to affect CD4+ T cells directly and not at the antigen-presenting
cells. These results therefore provide an addnl. pathway by which NO
modulates the immune response and contributes to the homeostasis of the
immune system.
REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 62 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 18
ACCESSION NUMBER: 1999:376749 CAPLUS
DOCUMENT NUMBER: 131:30804
TITLE: The mucosal adjuvant effects of cholera toxin and
immune-stimulating complexes differ in their
requirement for ***IL*** - ***12*** , indicating
different pathways of action
AUTHOR(S): Grdic, Dubravka; Smith, Rosemary; Donachie, Anne;
Kjerrulf, Martin; Hornquist, Elisabeth; Mowat, Allan;
Lycke, Nils
CORPORATE SOURCE: Department Medical Microbiology Immunology, Univ.
Goteborg, Goteborg, S-41346, Swed.
SOURCE: European Journal of Immunology (1999), 29(6),
1774-1784
CODEN: EJIMAF; ISSN: 0014-2980
PUBLISHER: Wiley-VCH Verlag GmbH
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Adjuvants that can improve mucosal vaccine efficacy are much warranted.
Studying cholera toxin (CT) and immune-stimulating complexes (ISCOM) the
authors found that, contrary to CT, ovalbumin (QVA)-ISCOM were poor
inducers of mucosal anti-OVA IgA responses, but induced similar or better
systemic immunity following oral immunizations. The addn. of CT to the
oral OVA-ISCOM protocol did not stimulate local anti-OVA IgA immunity, nor
did it change the quality or magnitude of the systemic responses. Both
vectors recruited strong innate immunity, but only OVA-ISCOM could
directly induce ***IL*** - ***12*** , demonstrable at the protein and
mRNA levels. CT had ***no*** ***inhibitory*** effects on
lipopolysaccharide/IFN-.gamma.-induced ***IL*** - ***12*** mRNA
expression or ***IL*** - ***12*** prodn. Adjuvanticity of CT was
unaffected in ***IL*** - ***12*** -deficient mice, while OVA-ISCOM
showed partly impaired adjuvant effects by the lack of ***IL*** -
12 . CT abrogated the induction of oral tolerance stimulated by
antigen feeding in these mice. CT did not alter TGF-.beta. levels,
suggesting that the immunomodulating effect of CT was independent of
IL - ***12*** as well as TGF-.beta. prodn. These findings

indicate that mucosal adjuvanticity of CT and ISCOM are differently
dependent on ***IL*** - ***12*** , suggesting that sep. and distinct
antigen-processing pathways are involved.
REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 63 OF 74 MEDLINE on STN DUPLICATE 19
ACCESSION NUMBER: 2000099817 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10634000
TITLE: Depressed cytolytic activity of peripheral blood
mononuclear cells in unusually high paclitaxel
concentrations: reversal by IL-2 and ***IL*** - ***12*** .
AUTHOR: Chen Y M; Yang W K; Ting C C; Yang D M; Whang-Peng J; Perng R P
CORPORATE SOURCE: Department of Chest Department, Taipei Veterans General Hospital, Taiwan, ROC.
SOURCE: Zhonghua yi xue za zhi = Chinese medical journal; Free China ed, (1999 Dec) 62 (12) 867-74.
Journal code: 0005327. ISSN: 0578-1337.
PUB. COUNTRY: China
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200001
ENTRY DATE: Entered STN: 20000209
Last Updated on STN: 20000209
Entered Medline: 20000128
AB BACKGROUND: Human lymphocyte function was inhibited by high concentrations
of paclitaxel and the effect was reversed by interleukin (IL)-2. However,
there was no parallel study determining the relationship between
paclitaxel concentrations in the lymphocyte cultures and pharmacokinetic
analysis in human patients, nor was there any study on the reversal by
cytokines, other than IL-2, of the paclitaxel-induced suppression of
lymphocyte cytotoxicity. METHODS: We tested the effect of different doses
of paclitaxel with various incubation times on the cytolytic activity of
peripheral blood mononuclear cells (PBMNCs) against K-562 target cells.
RESULTS: Our results showed that using a schedule similar to that for
treating patients with tolerable doses of paclitaxel, ***no*** ***inhibition*** of cytolytic activity of PBMNCs was seen. When the
paclitaxel concentration was increased 10-fold, the cytolytic activity of
PBMNCs was significantly reduced. This suppression was reversed by the
simultaneous addition of a low dose (10 U/ml) of IL-2 or ***IL*** - ***12*** . Addition of granulocyte macrophage-colony stimulating factor
(10 U/ml) did not affect the cytolytic activity of PBMNCs, whereas
addition of IL-4 reduced it. Time kinetic studies revealed that, with the
addition of IL-2 or ***IL*** - ***12*** , most of the mononuclear
cellular cytolytic activity recovered within 48 to 72 hours. CONCLUSIONS:
These findings suggested that, to reduce the toxicity on mononuclear
cellular function when high-dose paclitaxel treatment is elected in
clinical practice, paclitaxel should be infused over a longer duration of
time, or the treatment should be combined with the administration of a low
dose of IL-2 or ***IL*** - ***12*** .

L5 ANSWER 64 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 20
ACCESSION NUMBER: 1999:409846 CAPLUS
DOCUMENT NUMBER: 131:227568
TITLE: Mechanisms of Cytokine-Mediated Inhibition of Viral
Replication
AUTHOR(S): Komatsu, Takashi; Srivastava, Neil; Revzin, Margarita;
Ireland, Derek D. C.; Chesler, David; Shoshkes Reiss,
Carol
CORPORATE SOURCE: Department of Biology, New York University, New York
City, NY, 10003-6688, USA
SOURCE: Virology (1999), 259(2), 334-341
CODEN: VIRLAX; ISSN: 0042-6822
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Here, the role of nitric oxide synthase (NOS) and ***IL*** - ***12***
administration in inhibition of vesicular stomatitis virus (VSV) from
infected neuroblastoma cells was examd. The authors previously have shown
that cytokine treatment of cells results in the induction of NOS-1, and
this is assocd. with a 2 log inhibition of VSV prodn. The authors
performed these studies to examine the mechanism by which viral

replication is suppressed. Neuroblastoma cells (NB41A3) were treated with
either ***IL*** - ***12*** or medium and subsequently infected with
VSV. Viral protein and mRNA were isolated from these cells, and their
levels were measured by Western or Northern blots, resp. MRNA levels were
decreased modestly, but viral proteins were decreased substantially in
cells pretreated with ***IL*** - ***12*** , suggesting that the
inhibitory effect of NO is working at the translational level. Cytokine
treatment of cells was not assocd. with oxidative stress. The viral
proteins also were nitrosylated. Apparently, the mechanism of ***NO***
inhibition of viral replication occurs via translational
interference and posttranslational modifications of viral components. (c)
1999 Academic Press.
REFERENCE COUNT: 64 THERE ARE 64 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 65 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 21
ACCESSION NUMBER: 1998:800538 CAPLUS
DOCUMENT NUMBER: 130:51318
TITLE: Nitric oxide regulates Th1 cell development through
the inhibition of ***IL*** - ***12*** synthesis
by macrophages
AUTHOR(S): Huang, Fang-Ping; Niedbala, Wanda; Wei, Xiao-Qing; Xu,
Damo; Feng, Gui-Jie; Robinson, John H.; Lam, Charles;
Liew, Foo Y.
CORPORATE SOURCE: Department Immunology, University Glasgow, Glasgow,
G11 6NT, UK
SOURCE: European Journal of Immunology (1998), 28(12),
4062-4070
CODEN: EJIMAF; ISSN: 0014-2980
PUBLISHER: Wiley-VCH Verlag GmbH
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The authors have previously reported that mice lacking inducible NO
synthase (NOS2) developed enhanced Th1 cell responses. The authors now
investigated the mechanism by which NO modulates Th1 cells
differentiation. Peritoneal macrophages from NOS2-deficient mice infected
with Leishmania major in vivo or stimulated with interferon(IFN)-.gamma.
or lipopolysaccharide (LPS) in vitro produced higher levels of
interleukin(***IL***)- ***12*** than those from heterozygous or
wild-type mice. A macrophage cell line, J774, produced significant amts.
of ***IL*** - ***12*** following activation with LPS, or LPS +
IFN-.gamma.. This was markedly enhanced by the ***NOS***
inhibitor L-NG monomethyl Arg (L-NMMA), but profoundly inhibited
by the NO-generating compd. S-nitroso-N-acetyl-penicillamine (SNAP). The
effect of NO in this system is selective, since SNAP enhanced and L-NMMA
decreased TNF-.alpha. synthesis by LPS-activated J774 cells. The
differential effect of NO on ***IL*** - ***12*** and TNF-.alpha. is
at the transcriptional level and is activation dependent. Since
IL - ***12*** is a major inducer of Th1 cells which produce
IFN-.gamma. that can activate macrophages to produce ***IL*** -
12 , these data demonstrate that NO can be an inhibitor of this
feedback loop, preventing the excessive amplification of Th1 cells which
are implicated in a range of immunopathologies.
REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 66 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 22
ACCESSION NUMBER: 1998:717539 CAPLUS
DOCUMENT NUMBER: 130:80197
TITLE: Immune suppression by recombinant interleukin (rIL)-12
involves interferon .gamma. induction of nitric oxide
synthase 2 (iNOS) activity: inhibitors of NO
generation reveal the extent of rIL-12 vaccine
adjuvant effect
AUTHOR(S): Koblish, Holly Kurzawa; Hunter, Christopher A.;
Wysocka, Maria; Trinchieri, Giorgio; Lee, William M.
F.
CORPORATE SOURCE: Cell and Molecular Biology Graduate Group, Cancer
Center, and Institute for Human Gene Therapy, School
of Medicine, University of Pennsylvania, Philadelphia,
PA, 19104, USA

SOURCE: Journal of Experimental Medicine (1998), 188(9), 1603-1610
CODEN: JEMEAV; ISSN: 0022-1007
PUBLISHER: Rockefeller University Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Recombinant ***interleukin*** ***12*** (***IL*** - ***12***) can profoundly suppress cellular immune responses in mice. To define the underlying mechanism, recombinant murine (rm) ***IL*** - ***12*** was given to C57BL/6 mice undergoing alloimmunization and found to transiently but profoundly suppress in vivo and in vitro allogeneic responses and in vitro splenocyte mitogenic responses. Use of neutralizing antibodies and genetically deficient mice showed that IFN-.gamma. (but not TNF-.alpha.) mediated rmIL-12-induced immunosuppression. Splenocyte fractionation studies revealed that adherent cells from rmIL-12-treated mice suppressed the mitogenic response of normal nonadherent cells to Con A and IL-2. Addn. of an inhibitor of nitric oxide synthase (NOS) restored mitogenic responses, and inducible (i)NOS-/- mice were not immunosuppressed by rmIL-12. These results support the view that suppression of T cell responses is due to NO produced by macrophages responding to the high levels of IFN-.gamma. induced by rmIL-12. When a ***NOS*** ***inhibitor*** was given with rmIL-12 during vaccination of A/J mice with irradiated SCK tumor cells, immunosuppression was averted and the extent of rmIL-12's ability to enhance induction of protective antitumor immunity was revealed. This demonstrates that rmIL-12 is an effective vaccine adjuvant whose efficacy may be masked by its transient immunosuppressive effect.
REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 67 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 23
ACCESSION NUMBER: 1998:226196 CAPLUS
DOCUMENT NUMBER: 128:292200
TITLE: ***Interleukin*** ***12*** breaks ultraviolet light induced immunosuppression by affecting CD8+ rather than CD4+ T cells
AUTHOR(S): Schwarz, Agatha; Grabbe, Stephan; Mahnke, Karsten; Riemann, Helge; Luger, Thomas A.; Wysocka, Maria; Trinchieri, Giorgio; Schwarz, Thomas
CORPORATE SOURCE: Ludwig Boltzmann Institute for Cell Biology and Immunobiology of the Skin, Department of Dermatology, University Munster, Munster, D-48149, Germany
SOURCE: Journal of Investigative Dermatology (1998), 110(3), 272-276
CODEN: JIDEAE; ISSN: 0022-202X
PUBLISHER: Blackwell Science, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Recent studies showed that injection of interleukin (***IL***)-***12*** prevents UV light mediated suppression of contact hypersensitivity and breaks UV-induced hapten specific tolerance. UV-mediated suppression can be adoptively transferred by injecting splenocytes from UV-irradiated mice; however, suppression is not transferable when donor mice are treated with ***IL*** - ***12*** after UV-irradn. This study was performed to elucidate the mechanisms by which ***IL*** - ***12*** counteracts this immunosuppression. To characterize the cells transferring suppression, depletion studies were performed revealing that UV-induced suppression is transferred via CD8+ T cells. To investigate whether ***IL*** - ***12*** counteracts UV-induced suppression by either inhibiting the development of CD8+ suppressor T cells or inducing CD4+ effector T cells, splenocytes from mice, which were ***IL*** - ***12*** treated and sensitized through UV-exposed skin, were depleted from CD4+ T cells and transferred into naive mice that were subsequently sensitized. Whereas transfer of splenocytes from UV-irradiated mice inhibited sensitization of recipients, ***no*** ***inhibition*** was obsd. after transfer of splenocytes from UV-exposed and ***IL*** - ***12*** treated mice. Recipients that received CD4 depleted spleen cells from UV-exposed and ***IL*** - ***12*** treated donors, were still fully sensitizable. ***IL*** - ***12*** also blocked transfer of UV-induced suppression when it was

injected into UV-exposed donor animals at a time point when suppressor
cells had already developed. CD4 depletion of such splenocytes did not
result in a loss of the reconstitutive effect of ***IL*** - ***12***
. Thus, ***IL*** - ***12*** may break UV-induced tolerance not by
inducing CD4+ effector T cells, but rather by inhibiting or inactivating
suppressor T cells belonging to the CD8 subtype.
REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 68 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 24
ACCESSION NUMBER: 1998:698752 CAPLUS
DOCUMENT NUMBER: 130:37230
TITLE: Increased nitric oxide (NO) production by
antigen-presenting dendritic cells is responsible for
low allogeneic mixed leukocyte reaction (MLR) in
primary biliary cirrhosis (PBC)
AUTHOR(S): Yamamoto, K.; Akbar, Sk. Md. Fazle; Masumoto, T.;
Onji, M.
CORPORATE SOURCE: Third Department of Internal Medicine, Ehime
University School of Medicine, Ehime, 791-0295, Japan
SOURCE: Clinical and Experimental Immunology (1998), 114(1),
94-101
CODEN: CEXIAL; ISSN: 0009-9104
PUBLISHER: Blackwell Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The levels of blastogenesis in allogeneic MLR contg. T cells from one
normal volunteer and irradiated dendritic cells from 29 patients with PBC,
17 patients with chronic hepatitis type C (CH-C) and 22 allogeneic normal
controls were compared to see if there is any role of antigen-presenting
cells (APC) in the pathogenesis of PBC. The stimulatory capacity of
dendritic cells from PBC was lower than that of dendritic cells from CH-C
and normal controls, which could not be attributable either to the levels
of expression of surface mols., such as HLA-DR and CD86 on dendritic
cells, or to the levels of cytokines, such as IL-10 and ***IL*** -
12 . Higher levels of NO were seen in the allogeneic MLR
supernatants contg. dendritic cells from PBC compared with the
supernatants from cultures contg. dendritic cells from CH-C or normal
controls. Moreover, dendritic cells from PBC produced 10 times more NO
compared with dendritic cells from CH-C and normal controls. The addn. of
NG-monomethyl-L-arginine monoacetate (L-NMMA), a known inhibitor of NO in
allogeneic MLR contg. dendritic cells from PBC, resulted in a decrease of
NO and increase of blastogenesis. The selective impairment of dendritic
cell function, increased prodn. of NO by dendritic cells and restoration
of blastogenesis using ***NO*** ***inhibitor*** in PBC have
suggested a role for NO and dysfunction of dendritic cells in the
pathogenesis of PBC.
REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 69 OF 74 MEDLINE on STN DUPLICATE 25
ACCESSION NUMBER: 96304841 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8723796
TITLE: Inhibitory effects of ***interleukin*** ***12*** on
retroviral gene transduction into CD34 cord blood myeloid
progenitors mediated by induction of tumor necrosis
factor-alpha.
AUTHOR: Xiao M; Li Z H; McMahel J; Broxmeyer H E; Lu L
CORPORATE SOURCE: Department of Medicine (Hematology/Oncology), Indiana
University School of Medicine, Indianapolis, USA.
CONTRACT NUMBER: R01 HL46549 (NHLBI)
R01 HL54037 (NHLBI)
R37 CA36464 (NCI)
+
SOURCE: Journal of hematotherapy, (1996 Apr) 5 (2) 171-7.
Journal code: 9306048. ISSN: 1061-6128.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199610
ENTRY DATE: Entered STN: 19961022
Last Updated on STN: 19980206
Entered Medline: 19961009
AB ***Interleukin*** ***12*** (***IL*** - ***12***), a

heterodimeric cytokine with potent biologic activity, was evaluated for
effects on retroviral-mediated gene transduction into human myeloid
progenitor cells in vitro. Cord blood CD34 cells were prestimulated with
Steel factor (SLF), IL-3, GM-CSF, and erythropoietin (Epo) in the presence
and absence of 5-80 ng/ml ***IL*** - ***12*** for 40 hr in
suspension culture prior to gene transduction using viral supernatant
collected from a packaging cell line containing the pLNL6 vector encoding
Neo sequences. After gene transduction, cells were assayed for colony
formation stimulated by Epo, GM-CSF, IL-3, and SLF, and gene transduction
efficiency was determined by the percentage of G418 resistant (R) colonies
and confirmed by PCR analysis. ***IL*** - ***12*** dose-dependently
inhibited retroviral-mediated gene transduction into human cord blood CD34
granulocyte-macrophage (CFU-GM) and erythroid (BFU-E) progenitors. These
suppressive effects could be neutralized by incubation of ***IL*** -
12 with polyclonal antihuman ***IL*** - ***12*** .
IL - ***12*** had ***no*** ***inhibitory*** effects
directly on colony formation. To understand the possible mechanisms for
this suppression, ELISA assays were used to detect the release of
interferon (IFN)-gamma and tumor necrosis factor (TNF)-alpha, which could
potentially have been induced by ***IL*** - ***12*** from CD34
cells. TNF-alpha protein release was significantly increased in CD34
cells incubated with ***IL*** - ***12*** . No detectable levels of
IFN-gamma were noted. Anti-TNF-alpha, but not anti-IFN-gamma, blocked the
inhibitory effects of ***IL*** - ***12*** on gene transduction.
Moreover, TNF-alpha, but not IFN-gamma, suppressed gene transfer to the
same degree as ***IL*** - ***12*** . No change of amphotropic
receptor mRNA expression was noted by Northern blot analysis in cells
treated with or without ***IL*** - ***12*** . The results suggest
that the suppressive effects of ***IL*** - ***12*** on retroviral
gene transduction are, at least in part, mediated by ***IL*** -
12 induction of the release of TNF-alpha.

L5 ANSWER 70 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 26
ACCESSION NUMBER: 1996:507756 CAPLUS
DOCUMENT NUMBER: 125:193084
TITLE: Interferon-.gamma. induced type I nitric oxide
synthase activity inhibits viral replication in
neurons
AUTHOR(S): Komatsu, Takashi; Bi, Zhengbiao; Reiss, Carol S.
CORPORATE SOURCE: Department of Biology, New York University, New York,
NY, 10003, USA
SOURCE: Journal of Neuroimmunology (1996), 68(1-2), 101-108
CODEN: JNRIDW; ISSN: 0165-5728
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Type I NOS expression increases in OB neurons during VSV infection.
Immunocytochem. staining of NB41A3 cells indicates constitutive expression
of interferon (IFN)-.gamma. receptor and type I NOS. IFN-.gamma.
treatment of NB41A3 cells increased NO prodn. and type 1 NOS protein. In
vitro replication of VSV, polio virus type 1, and Herpes Simplex virus
type 1 (HSV-1) is significantly inhibited by IFN-.gamma. induced type I
NOS and antagonized by ***NOS*** ***inhibitors*** . In contrast,
while IFN-.gamma. treatment inhibited influenza and Sindbis virus
replication, a different pathway(s) was involved. The isoform-selective
NOS ***inhibitor*** , 7-nitroindazole (7NI) was used to treat
mice, resulting in a 10-fold higher titer of virus in brain homogenates,
and abrogated the recovery-promoting effect of ***interleukin*** -
12 treatment. Thus, IFN-.gamma. induced type I NOS activity may
play an important role in host immunity against neurotropic viral
infections.

L5 ANSWER 71 OF 74 MEDLINE on STN
ACCESSION NUMBER: 96197369 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8625365
TITLE: Effects of N(g)-methyl-L-arginine, an inhibitor of nitric
oxide synthesis, on interleukin-2-induced capillary leakage
and antitumor responses in healthy and tumor-bearing mice.
AUTHOR: Orucevic A; Lala P K
CORPORATE SOURCE: Department of Anatomy, University of Western Ontario,

Canada.
SOURCE: Cancer immunology, immunotherapy : CII, (1996 Jan) 42 (1) 38-46.
Journal code: 8605732. ISSN: 0340-7004.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199606
ENTRY DATE: Entered STN: 19960708
Last Updated on STN: 19960708
Entered Medline: 19960626

AB We tested whether treatment with an inhibitor of nitric oxide synthesis (N g -methyl-L-arginine, MeArg) can ameliorate interleukin-2(IL-2)-therapy-induced capillary leak syndrome in healthy or tumor-bearing mice without compromising the antitumor effects of IL-2 therapy. Healthy or C3-L5-mammary-adenocarcinoma-bearing C3H/HeJ mice were treated with one or two rounds of various doses of IL-2 (ten injections, i. p., every 8 h) or MeArg (ten injections s. c., every 8 h) or their combination. In an additional experiment, MeArg was given chronically in the drinking water, rather than s. c. to healthy mice subjected to one round of therapy as above. Mice were killed 1 h after their last IL-2 injection to measure the water content of the lungs and pleural cavities (markers of capillary leakage), NO production (given by NO2- and NO3- levels in the serum and pleural effusion), as well as the effect of therapies on the primary tumor size and number of spontaneous lung metastatic nodules. Results revealed that all doses of IL-2 (7500-35000 Cetus U/injection), as well as both rounds of IL-2 therapy, caused capillary leakage. However, no pleural effusion was seen after the second round in any of the IL-2-treated groups. MeArg therapy, given subcutaneously (5-20 mgkg(-1) injection(-1) in healthy and 20 mgkg(-1) injection(-1) in tumor-bearing mice), did not ameliorate IL-2-induced capillary leakage in either group of mice, and did not compromise antitumor effects of IL-2. However, subcutaneous MeArg therapy alone reduced the growth of the primary tumors, the occurrence of lung metastases and the amount of tumor-induced pulmonary edema. When MeArg therapy was given orally (1 mg/ml drinking water), a substantial drop in NO production, as well as reduction in capillary leakage was noted in IL-2-treated healthy mice. These findings suggest that ***NO*** ***inhibitors*** could be a valuable adjunct to IL-2 therapy of cancer and infectious diseases.

L5 ANSWER 72 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 27
ACCESSION NUMBER: 1995:470536 CAPLUS
DOCUMENT NUMBER: 122:237476
TITLE: ***Interleukin*** - ***12*** profoundly up-regulates the synthesis of antigen-specific complement-fixing IgG2a, IgG2b and IgG3 antibody subclasses in vivo
AUTHOR(S): Germann, Tieno; Bongartz, Martina; Dlugonska, Henryka; Hess, Henry; Schmitt, Edgar; Kolbe, Ludger; Koelsch, Eckehart; Podlaski, Frank J.; Gately, Maurice K.; Ruede, Erwin
CORPORATE SOURCE: Institute fuer Immunologie, Mainz, D-55101, Germany
SOURCE: European Journal of Immunology (1995), 25(3), 823-9
CODEN: EJIMAF; ISSN: 0014-2980
PUBLISHER: VCH
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The influence of the cytokine ***interleukin*** - ***12*** (***IL*** - ***12***) on humoral immune responses was studied in vivo. CBA/J mice immunized with protein antigens (keyhole limpet hemocyanin, phospholipase A2) adsorbed to aluminum hydroxide (Alum) develop a Th2-like immune response characterized by the prodn. of large amts. of IgG1 as well as some IgE but little IgG2b and IgG3 antibodies. ***IL*** - ***12*** is a cytokine that promotes the development and the activation of Th1 cells. Th1 cells are involved in the induction of cellular immunity, which is characterized by low or absent antibody prodn. Some Th1-like immune responses are assocd. with a strong antibody prodn. of the IgG2a, IgG2b, and IgG3 subclasses. Thus, the authors investigated whether treatment with ***IL*** - ***12*** would down-regulate the humoral immune response to stimulate antibody prodn. of the IgG2a, IgG2b and IgG3

subclasses. The authors obsd. that administration of ***IL*** - ***12*** to mice together with protein antigens adsorbed to Alum strongly enhanced the humoral immune response by increasing the synthesis of antigen-specific antibodies of the IgG2a, IgG2b and IgG3 subclasses 10- to 1000-fold. The synthesis of IgG1 was not or only slight (2-5-fold) enhanced, whereas that of the IgE isotype was suppressed. These effects of ***IL*** - ***12*** were obsd. when high (10 .mu.g, 100 .mu.g) or low doses (0.1 .mu.g) of antigen were used for immunization. Titrn. of ***IL*** - ***12*** in vitro revealed that IgG2a is strongly up-regulated over a wide dose range of ***IL*** - ***12*** (10 to 1000 ng/day). The effects of ***IL*** - ***12*** in vivo are at least partially interferon (IFN)-.gamma.-dependent because an anti-IFN-.gamma. mAb in combination with ***IL*** - ***12*** prevented most of the enhanced IgG2a prodn. Mice receiving ***IL*** - ***12*** showed a strong up-regulation of IFN-.gamma. but ***no*** ***inhibition*** of IL-5 synthesis by spleen cells activated ex vivo with antigen. There results suggest that ***IL*** - ***12*** is a potent adjuvant for enhancing humoral immunity to protein antigens adsorbed to Alum, primarily by inducing the synthesis of the complement-fixing IgG subclasses 2a, 2b, and 3.

L5 ANSWER 73 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 28
ACCESSION NUMBER: 1995:330082 CAPLUS
DOCUMENT NUMBER: 122:103698
TITLE: Mouse ***interleukin*** - ***12*** (***IL*** - ***12***) p40 homodimer: a potent ***IL*** - ***12*** antagonist
AUTHOR(S): Gillessen, Silke; Carvajal, Daisy; Ling, Ping; Podlaski, Frank J.; Stremlo, Donna L.; Familletti, Philip C.; Gubler, Ueli; Presky, David H.; Stern, Alvin S.; Gately, Maurice K.
CORPORATE SOURCE: Dep. Inflammation/Autoimmune Dis., Hoffman-La Roche Inc., Nutley, NJ, 07110, USA
SOURCE: European Journal of Immunology (1995), 25(1), 200-6
CODEN: EJIMAF; ISSN: 0014-2980
PUBLISHER: VCH
DOCUMENT TYPE: Journal
LANGUAGE: English
AB ***Interleukin*** - ***12*** (***IL*** - ***12***) is a cytokine that has regulatory effects on T and natural killer (NK) cells and is composed of 2 disulfide-bonded subunits, p40 and p35. It was recently reported that supernatants from cultures of mouse ***IL*** - ***12*** (moIL-12) p40-transfected COS cells could inhibit ***IL*** - ***12*** -dependent responses in vitro (Mattner, F., et al., 1993). The authors have further characterized the nature of the inhibitory substance. Purified mouse p40 produced in a baculovirus expression system was found to consist of 2 species: the p40 monomer and a disulfide-linked p40 dimer [(p40)2]. The (p40)2 was 25-50-fold more active than the p40 monomer in causing specific, dose-dependent inhibition of ***IL*** - ***12*** -induced mouse Con A blast proliferation and also inhibited ***IL*** - ***12*** -induced interferon-.gamma. (IFN-.gamma.) secretion by mouse splenocytes and ***IL*** - ***12*** - dependent activation of mouse NK cells. Competitive binding studies on mouse Con A blasts showed that (p40)2 was equally effective as moIL-12 in competing with 125I-labeled moIL-12 ([125I]moIL-12) for binding to mouse Con A blasts. However, in contrast to moIL-12, mouse (p40)2 displayed little ability to compete with 125I-labeled human ***IL*** - ***12*** (huIL-12) for binding to high-affinity ***IL*** - ***12*** receptors (IL-12R) on human phytohemagglutinin (PHA) blasts and caused little or ***no*** ***inhibition*** of huIL-12-induced human PHA blast proliferation. Nonetheless, mouse (p40)2 was equally effective as moIL-12 in competing with [125I]huIL-12 for binding to COS cells transfected with the human IL-12R .beta. subunit and expressing low-affinity ***IL*** - ***12*** binding sites. Thus, (1) the majority of the structural determinants required for binding of ***IL*** - ***12*** to its receptor are contained within the p40 subunit, but p35 is required for signaling, (2) the p40 subunit of ***IL*** - ***12*** interacts with the .beta.

subunit of IL-12R, and (3) (p40)2 may be a suitable ***IL*** -
12 antagonist for studying the role of ***IL*** - ***12***
in various immune responses in vivo as well as in vitro.

L5 ANSWER 74 OF 74 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 29
ACCESSION NUMBER: 1994:189371 CAPLUS
DOCUMENT NUMBER: 120:189371
TITLE: Differential effects of ***interleukin*** -
12 on the development of naive mouse CD4+ T
cells
AUTHOR(S): Schmitt, Edgar; Hoehn, Petra; Germann, Tieno; Ruede,
Erwin
CORPORATE SOURCE: Inst. Immunol., Mainz, D-55101, Germany
SOURCE: European Journal of Immunology (1994), 24(2), 343-7
CODEN: EJIMAF; ISSN: 0014-2980
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The influence of interleukin (***IL***)-***12*** and IL-4 on the
differentiation of naive CD4+ T cells was studied in an accessory
cell-free in vitro system. Dense CD4+ T cells were purified from
unimmunized mice and activated using immobilized anti-CD3 monoclonal
antibodies (mAb) in the presence of IL-4, ***IL*** - ***12*** , or a
combination of both cytokines, and restimulated after 6 days by
re-exposure to anti-CD3-coated culture wells. T cells initially activated
in the presence of IL-4 produced substantial amts. of IL-4 and trace amts.
of interferon (IFN)-.gamma. after restimulation at day 6 with plate-bound
anti-CD3 mAb. By contrast, T cells primed in the presence of ***IL***
- ***12*** produced high levels of IFN-.gamma. and only minimal amts.
of IL-4, thus indicating that IL-2 and IL-4 by acting directly on
stimulated naive CD4+ T cells support the development of TH1 and TH2
cells, resp. When naive CD4+ T cells were stimulated in the presence of
IL - ***12*** together with IL-4 in comparable concns., the
effect of ***IL*** - ***12*** on TH1 differentiation was largely
inhibited by IL-4. ***IL*** - ***12*** exerted ***no***
inhibitory effect on IL-4-induced TH2 differentiation but rather
enhanced the prodn. of IL-4 after restimulation of the resp. T cells.
Decreasing amts. of IL-4 in combination with a high level of ***IL*** -
12 led to an increasing prodn. of IFN-.gamma. by the emerging T
cells and, simultaneously, to a relatively high prodn. of IL-4. These
data were confirmed by time-course expts. which revealed that the delayed
addn. of IL-4 to ***IL*** - ***12*** -primed T cell cultures resulted
in a gradual restoration of IFN-.gamma. prodn. whereas in parallel the
secretion of IL-4 was not reduced over a wide period of delay (6-72 h).
These results, therefore, demonstrate that (a) IL-4 dominates the effect
of ***IL*** - ***12*** , (b) ***IL*** - ***12*** promotes the
development of TH1 cells; however, in the presence of ***IL*** -
12 and relatively high levels of IL-4 also the development of
TH2-like cells is slightly enhanced by ***IL*** - ***12*** , and (c)
high amts. of ***IL*** - ***12*** in combination with relatively low
levels of IL-4 give rise to a T cell population that upon rechallenge
exhibited a cytokine profile resembling that of TH0 cells.

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ENTRY SESSION